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One of the two tubercles found in the wall of the colon was imbedded in paraffin and afterwards cut and microscopically examined. It was thus ascertained that the crater-like excavation was the result of a loss of tissue determined by suppuration, which extended beneath the muscularis mucosæ. At the base of the ulcer there were many leucocytes, and chromatin granules which stained blue with hæmatoxylin. The part of the glandularis mucosæ adjacent to the ulcer was beset with round cells and leucocytes. Close to the ulcer the gland tubes had been almost or entirely destroyed, but a little further away from the ulcer the gland tubes were still recognisable, and one could make out that the process had had its starting-point in the submucosa. Many leucocytes had collected between the gland tubes and in their interior, and the gland cells were detached from the membrana propria of the glands. Many of the gland cells are destroyed, only the nuclear chromatin remaining in the form of small globules and droplets. The goblet cells have resisted the longest, and their form can be made out even at places where the other elements have been liquefied. Above the muscularis mucosæ one can also recognise eosinophilous cells, which are either collected together in nests or arranged in rows. Rows of these cells are also found in the submucosa. Round cells and leucocytes are, however, found much more abundantly in the submucosa, where they have formed small abscesses which are limited by the muscularis mucosæ. The latter has prevented the abscesses from breaking through into the glandularis mucosæ. It is precisely this occurrence of the abscesses in the submucosa which indicates that the breaking-down process had its beginning there, and afterwards extended to the muscularis mucosæ and glandularis mucosæ. Even in the crater-like ulcer of the tubercle it can still be made out that the degeneration of the latter had its starting-point in the submucosa.

2. On the forenoon of the 28th June 1896, a gelding, twelve years old, was given a potato ball surrounded by gelatine and containing the glanders bacilli furnished by sixteen potato and twenty-four glycerine agar cultures. The cultures of glanders bacilli had been obtained by inoculation from pieces of the glanderous mesenteric lymphatic gland of horse No 1. The horse swallowed the ball quite well. The temperature of the horse after the administration of the ball is shown in the following table :

[TABLE.

Day.	Forenoon.	Noon.	Afternoon.	
28th June .	37˙5	37˙6	37˙8	
29th „ .	37˙5	37˙6	37˙7	
30th „ .	39˙8	40˙1	40˙1	⎫ Pulse 56 and respirations
1st July .	39˙9	39˙3	40˙1	⎬ 12 per minute. No ⎭ appetite ; dull.
2nd „ .	39˙5	39˙1	39˙1	
3rd „ .	39˙0	39˙1	39˙2	Slight appetite.
4th „ .	39˙5	39˙6	39˙7	
5th „ .	39˙2	39˙3	38˙7	Slight appetite.
6th „ .	39˙2	39˙2	39˙5	
7th „ .	39˙1	39˙0	38˙8	
8th „ .	38˙8	38˙9	38˙7	
9th „ .	39˙9	39˙3	39˙1	

In this horse also the body temperature rose during the course of the second day, and afterwards remained at about the same height.

On the evening of the 9th July the horse was injected with Preusse's mallein. The result of the test was as follows ;

Day.	12 Midnight.	6 a.m.	8 a.m.	10 a.m.	12 noon.	2 p.m.	4 p.m.	6 p.m.	8 p.m.
9th July .	39˙6	—	—	—	—	—	—	—	—
10th „ .	—	37˙9	38˙2	38˙5	38˙8	38˙6	38˙4	38˙3	38˙3
11th „ .	—	38˙0	—	—	—	—	—	—	—

The injection of the mallein was thus not followed by any reaction. On the 11th July the horse was killed, and the *post-mortem* examination disclosed the following :—

Post-mortem Record.—Body moderately well nourished. Rigor mortis not yet set in. The eyelids are open. The anus is closed. The conjunctival membrane is pale red. At the seat of injection, on the left side of the neck, the subcutaneous tissue is saturated with bloody fluid. With this exception the skin and subcutaneous tissue are normal. The peritoneal cavity contains a quarter of a litre of yellowish clear fluid. The position of the abdominal organs is normal. The peritoneum is pale red and shining. The contents of the stomach are dry, those of the small intestine fluid, and those in the large intestine pultaceous or dry. In the pyloric part of the stomach there is a 2 cm. long and 0˙5 cm. broad cicatrix, the edges and wall of which are smooth and thick, while its base is reddened. In the wall of the small intestine one can feel two pea-sized tubercles which have their seat in the submucosa, and appear grey through the unaltered mucous

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membrane ; a third tubercle of similar size is found in the submucosa of the large colon. In the submucosa of the point of the cæcum there are two flat thickenings about the size of a penny piece ; the mucous membrane over these is reddened, the reddening being also observable on the serous covering of the cæcum. These are the only alterations recognisable in the mucous membrane of the alimentary canal.

The whole of the mesenteric lymphatic glands are enlarged. The lymphatic glands in immediate contact with the intestines are mostly about the size of a pea, but those next the vertebral column, around the roots of the mesenteries, are the size of a hazel-nut. The glands are in part grey and firm, in part pale yellow and soft, and beset with greyish-yellow, opaque, dry, degenerated areas about the size of a pin's head. The lymphatic vessels of the mesentery form grey tortuous streaks of the thickness of a linen thread.

In the spleen, immediately under its capsule, there are about twelve miliary greyish-yellow tubercles with softened centres. In the liver there are occasional greyish-white tubercles of the size of a grain of oatmeal ; commencing softening can be observed in the centre of these.

The kidneys are normal.

The inguinal, iliac, pubic, and sacral lymphatic glands are greyish-red, swollen, and œdematous.

The pleural cavity contains a tablespoonful of clear yellowish fluid. The lungs are bright red, and contain air. Many grey opaque tubercles, varying in size from a pea to a hazel-nut, are distributed partly underneath the pleura, and partly in the lung substance. The tubercles under the pleura project a little above the surface of the lung. The small pea-sized tubercles are for the most part greyish-white, opaque, and beset with pinhead greyish-yellow points, most of which are surrounded by a thin red zone. In the case of the tubercles of the size of a bean or hazel nut the cut surface has at the periphery a grey, smooth, opaque appearance, and shows at the centre yellowish areas about the size of a pin's head. The lung tissue surrounding the tubercles is reddened and œdematous. In the anterior lobe of the left lung there are two firm areas as large as a hen's egg, and of a glassy, yellowish-red appearance ; on section one of these nodules shows a uniformly moist, greyish-red, smooth surface, while the other is at its centre granular, yellow, greyish-yellow, or greyish-red, and opaque. An area of the same character and as large as the fist is present in the posterior lobe of the left lung. The mediastinal lymphatic glands are as large as a hazel nut, the bronchial lymphatic glands as large as a walnut, and markedly œdematous.

The heart shows no lesions. The mucous membrane of the nose, pharynx, larynx, trachea, and bronchi is normal.

The brain is also normal.

From the degenerated masses in the mesenteric lymphatic glands cover-glass preparations were prepared, and in these glanders bacilli were detected. Furthermore, numerous colonies of glanders bacilli developed on potatoes and glycerine agar which had been inoculated with degenerated material from the lymphatic glands, and two guinea-pigs which were inoculated with that material became glandered.

The pea-sized tubercle in the submucosa of the large colon was found by microscopic examination to be an abscess, composed

of leucocytes, round cells, and remains (chromatin particles) of disintegrated cells. The part of the intestinal wall adjoining the abscess, especially the submucosa, is infiltrated with leucocytes. The tissue of the mucosa between the glands is filled with leucocytes and other cells resulting from proliferation of the fixed cells. The capillary vessels are also markedly filled with blood. Furthermore, many leucocytes are present in the lumen of the glands, and between the glandular epithelium. At several places the glands have begun to break down. The degeneration is further advanced in the mucosa, and has thus spread out from the depth towards the surface.

Similar alterations are present in the flat thickenings of the mucous membrane of the point of the cæcum, but the purulent liquefaction of the tissues is confined to the submucosa, whereas the glandularis mucosæ is still completely intact. The breaking down of the tissue along the plane of the mucosa is striking in the flat swollen parts of the point of the cæcum.

In both horses, besides the lymphatic glands, the chyle vessels in the great mesentery were also diseased. Whereas in healthy horses the chyle vessels of the small intestine appear in the form of yellowish-white lines running between the small intestine and the root of the mesentery, those in the experimental horses were very wide and strikingly white, thus appearing as if they were injected with milk. The chyle vessels show a similar appearance in tuberculosis of the human subject, or in cattle, when the intestine and the mesenteric lymphatic glands are affected. The result of the microscopic examination of the glandered chyle vessels is important.

The wall of the strikingly white chyle vessels is very thick ; the thickening is determined by the proliferation of the intima, especially of its endothelium. The intima shows a lamellated structure, and at some places the lamellæ of stratified endothelium is continued in the form of branched processes into the lumen of the chyle vessel. At these places the lumen is divided into areas which are composed of endothelial masses lying side by side. These fields represent the endothelial processes projecting into the interior of the chyle vessels, and their appearance is different according as they are cut in the longitudinal or in the transverse direction. When they are cut in the longitudinal direction the endothelium shows a sheaf-like arrangement, such as one can often recognise in crystals of the fatty acids. When the projections are cut in a transverse direction one sees the endothelium on the flat, and it appears like flat epithelial cells applied one against the other. Hence, in the contents of these chyle vessels one recognises a structure similar to that of an endothelioma. The passage for the stream of chyle is limited to the semilunar space which is left between the proliferated masses of endothelium and the wall of the chyle vessels, and to the spaces which are recognisable between the endothelial processes. The rate of flow of the chyle is thus reduced, and the chyle accumulates in the chyle vessels, in consequence of which the pressure exerted on the inner surface of the vessel rises, and the vessel becomes dilated. This pressure of the chyle also explains why the endothelial processes which fill up the lumen of the chyle vessel show a close texture.

Glanders bacilli are found between the proliferated endothelial cells. On the other hand, migrated white blood corpuscles cannot be

recognised either in the wall of the chyle vessel or in the endothelium. In all the endothelial cells the nucleus is quite discernible, and there is no trace anywhere of degeneration of the endothelium.

It has accordingly been proved that a glanderous infection may be produced by feeding with large quantities of glanders bacilli in the manner above described. A striking fact in this connection is that the mucous membrane of the small and large intestines showed very slight glanderous lesions, whereas the chyle vessels and the mesenteric lymphatic glands were severely affected. This is analogous to those cases of tuberculosis of the mesenteric glands in cattle in which the intestine is either quite free or almost free from tuberculous lesions. But it has not been proved that the lungs are primarily affected with glanders when the glanders bacilli are introduced into horses by way of the alimentary canal, since in both the experimental horses the chyle vessels and the mesenteric lymphatic glands as well as the intestine were the seat of glanderous lesions, and the lungs were thus the secondary seat of the disease. A still more striking fact is that in neither of the experimental horses were grey translucent tubercles to be found in the lungs, although the horses had been killed fourteen and thirteen days respectively after the administration of the ball.

It is true, however, that Nocard in the article referred to does not distinctly state that the grey translucent tubercles which developed in the manner above described are to be regarded as the product of primary pulmonary glanders. But this conclusion is in harmony with his earlier views, and other authors are also of the opinion that primary glanders of the lung can develop as the result of an infection with glanders bacilli, by way of the digestive apparatus. Kitt, for example, expresses himself to this effect.[1]

" The cause of development of primary glanders in the lung, however, is quite original, and it was recently explained for the first time through the genius of Nocard. This distinguished investigator in the domain of veterinary medicine has experimentally followed up the transmissibility of glanders by feeding, and by a series of exact experiments has strictly proved that a primary embolic glanders of the lung is brought about by feeding with glanders bacilli, which pass through the intestinal wall or the chyle vessels, and, apparently by way of the thoracic duct and anterior vena cava, are carried to the lungs. The paths traversed by the bacilli remain free from glanders lesions, and the lungs first furnish the soil for the vegetation of the bacilli which by this roundabout way have gained the smaller circulation."

Putting aside the fact that both the results of the preceding experiments and those of the experiments hereafter to be described teach the contrary, this conclusion cannot be regarded as justified by Nocard's views concerning the observations which he made on his experimental horses, for, as has already been mentioned, Nocard admits that in " almost all " the experimental horses to which glanders bacilli were administered in carrots or in drinking water the submaxillary lymphatic glands were simultaneously attacked, and that, indeed, in some of the horses actual ulcers of the mucous membrane of the nose were produced. In the absence of an account of the *post-mortem* examina-

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[1] Kitt, Lehrbuch der pathologisch-anatomischen Diagnostik II., S. 289.

tions, it would thus appear much more justifiable, or at least as justifiable, to refer the tubercles which Nocard found in the lung to a glanderous process of the upper part of the digestive apparatus, as to regard them as evidence of primary pulmonary glanders. With regard to this point, an experiment in which the method selected by Nocard for administering the glanders bacilli was accidentally imitated is quite conclusive.

3. On the forenoon of the 28th June 1896, it was intended to give to a fourteen-year-old mare two potato balls in which were included the glanders bacilli grown on sixteen potato and twenty-four glycerine agar cultures. The first ball was swallowed by the mare all right, but the other, while being pushed into the pharynx, broke at the back of the tongue, so that the contents of the ball, that is to say, the glanders bacilli, came into contact with the mucous membrane of the mouth and fauces. The cultures of glanders bacilli had been grown on potatoes and glycerine agar that had been inoculated from parts of the glandered mesenteric glands of horse No. 1, and their virulence had been proved by the inoculation of guinea-pigs. After the second ball had been broken in the act of administration the mare was offered water, of which she drank a large quantity. The temperature of the mare before and after the administration of the balls is shown in the following table:—

Day.	Forenoon.	Noon.	Evening.
	°	°	°
28th June . .	—	—	38·1
29th ,, . .	37·7	37·9	37·7
30th ,, . .	38·6	40·0	40·6
1st July . .	39·8	39·4	39·9
2nd ,, . . .	39·3	40·7	40·1
3rd ,, . . .	38·8	39·3	39·8
4th ,, . . .	39·7	39·6	39·3
5th ,, . . .	38·8	39·3	39·8
6th ,, . . .	38·9	38·9	39·1
7th ,, . . .	39·1	39·3	39·7
8th ,, . . .	39·2	39·2	39·9
9th ,, . . .	39·4	39·6	39·8

It will be seen from the above that an elevation of temperature was recognisable on the second day after the administration of the balls, and that the temperature subsequently never fell as low as it was before the beginning of the experiment. On the 30th June strong rigors and nasal discharge were observed in the mare, and on the 1st July cough and swelling of the submaxillary lymphatic glands set in. By the 9th July the lymphatic glands had attained the size of a

pigeon's egg and they remained painful. At the same time the respirations were increased to 126 in the minute.

On the evening of the 9th July ·5 gramme of Preusses' mallein was injected into the animal. The effect of the injection on the body temperature was as follows :—

Day.	12 Mid-night.	6 a.m.	8 a.m.	10 a.m.	12 noon.	2 p.m.	4 p.m.	6 p.m.	8 p.m.
9th July .	39·8	—	—	—	—	—	—	—	—
10th ,, .	—	38·6	39·2	39·5	39·6	39·6	40·2	40·4	39·3
11th ,, .	—	39·3	—	—	—	—	—	—	—

No swelling formed at the seat of injection, and the above figures show that the body temperature remained unaltered after the injection of the mallein.

The mare was killed on the 12th July, and the *post-mortem* examination revealed the following :—

Post-mortem Record.—General condition pretty good. Rigor mortis not yet set in. Around the seat of injection in the neck the sub-cutaneous tissue is gelatinous and reddened. Some greyish-red turbid fluid flows from the nostrils.

The peritoneal cavity contains a small quantity of yellowish turbid liquid. The position of the intestines is normal. The peritoneum is smooth. The contents of the small intestine are fluid. In the submucosa of the small intestine, in a swollen part of the extent of a ten pfennig piece, there are numerous grey, opaque tubercles, of the size of a pin's head. One of these tubercles, which is seated directly under the glandularis mucosæ and softened at its centre, stands in communication with the lumen of the intestine; the mucous membrane at this place is reddened. The contents of the double colon and cæcum are semi-fluid; the mucous membrane of these intestines is normal. The contents of the floating colon and rectum are dry and in the form of balls; the mucous membrane normal. The contents of the stomach are small in quantity and semi-fluid; the mucous membrane of the stomach and œsophagus is normal. All the mesenteric lymphatic glands are enlarged, those on the intestine being the size of a pea and those around the root of the mesentery as large as a hazel nut and firm. On section the lymphatic glands are white or greyish-white, and show numerous pin-head, yellow, opaque, dry areas in process of degeneration. The chyle vessels are as thick as a linen thread and white.

In the liver there are greyish-white tubercles of the size of a grain of oatmeal and surrounded by a thin red zone. Some of the tubercles show purulent softening of their centres.

Spleen and kidneys normal.

The iliac, sacral, pubic, and inguinal glands are as large as beans, greyish-red, and œdematous.

The pleural cavities contain a small quantity of yellowish clear fluid. The lungs are pale red and contain air. The sub-pleural and interstitial tissue of the anterior lobe and the inferior middle part of the right lung are filled with rows of air bubbles. Under the

pulmonary pleura or in the substance of both lungs there are many tubercles varying in size from a grain of oatmeal to a bean. The smaller tubercles are outwardly greyish-white and opaque, in the centre yellow, and surrounded by a thin reddened zone. The larger tubercles contain pin-head yellow points of degeneration, in a grey tissue which has a granular cut surface. These degenerated points are surrounded by a moist reddened zone. In the posterior part of the right lung there is an amber yellow, airless, and very moist area of the size of a hen's egg ; on section this presents a greyish-red, opaque, granular, surface.

The mucous membrane of the larynx and trachea is pale. In the mucous membrane of the pharynx there are eight lentil-sized ulcers with abrupt edges. An ulcer of similar appearance is present on the aryteno-epiglottic fold. The single packets of the submaxillary and pharyngeal lymphatic glands vary in size from a bean to a hazel nut ; their cut surface is grey, moist, and beset with pin-head greyish-yellow spots. In some tubercles these spots have a whitish opaque appearance ; others are only juicy.

In cover-glass preparations from the degenerated areas in the sub-maxillary and mesenteric lymphatic glands very many glanders bacilli were recognisable, and pure cultures of these could be obtained by inoculation of potato and glycerine agar from the degenerated parts in the lymphatic glands. Lastly, two guinea-pigs which were inoculated with matter from degenerated areas became glandered.

This experiment shows that when horses take in glanders bacilli with the food or drinking water the upper part of the alimentary tube may also become infected with glanders, and that one may in this way obtain a result which Nocard observed in " almost all " his experimental horses. Such cases, however, do not indicate whether any glandered condition that may be found in the lungs had its origin in an infection with glanders bacilli from the intestine or pharynx. On the contrary, both the above described experiments leave no doubt that the intestine or the lymphatic glands lying in its neigh-bourhood may form primary seats for glanderous lesions in the lungs.

After it had thus been ascertained that the intestine can serve as the port of entrance for glanders bacilli when large quantities of these are administered to the horse, experiments were carried out to ascertain whether an infection could also be determined by the admin-istration of small quantities of bacilli. In the first instance a small quantity of glanders bacilli was on a single occasion given to a horse, and afterwards a second horse was repeatedly given the same quantity of bacilli.

4. On the 20th July 1896 the tenth part of a loopful of a glycerine agar culture of glanders bacilli, cultivated from a lymphatic gland of horse No 1, was rubbed up with 1 gramme of potato, and this was surrounded with gelatine so as to make a ball of the size of a small hen's egg. On the before-mentioned day this ball was administered to a twenty-year-old gelding. The temperature of the horse after-wards is shown in the following table :

[TABLE.

Day.	Forenoon.	Noon.	Evening.
21st July	37·6	37·8	38·0
22nd ,,	37·9	38·2	38·4
23rd ,,	37·9	37·9	37·8
24th ,,	37·7	37·8	37·9
25th ,,	37·3	37·8	37·5
26th ,,	37·4	37·8	37·9
27th ,,	37·5	37·7	37·7
28th ,,	37·4	37·6	38·9
29th ,,	37·6	37·7	37·7
30th ,,	37·6	37·7	37·6
31st ,,	37·5	37·7	38·0
1st August	37·3	37·5	37·8
2nd ,,	37·2	37·3	37·5
3rd ,,	37·2	37·6	37·4
4th ,,	36·8	37·3	37·6
5th ,,	37·7	37·7	37·6
6th ,,	37·0	37·6	37·4
7th ,,	37·2	37·4	37·8
8th ,,	37·3	37·7	37·7
9th ,,	37·1	37·3	37·5
10th ,,	36·9	37·4	37·4
11th ,,	36·6	37·6	37·5
12th ,,	36·1	37·6	37·9
13th ,,	37·9	37·2	37·4
14th ,,	37·1	37·4	37·6

It will be observed that no elevation of temperature was observable after the administration of the ball, nor was there any noticeable disturbance of the general health.

At midnight on the 14th August the horse received an injection of 1·5 ccm. of Preusse's mallein. The temperature afterwards is shown below :

Day.	Midnight.	6 a.m.	8 a.m.	10 a.m.	Noon.	2 p.m.	4 p.m.
14th August	37·8	—	—	—	—	—	—
15th ,,	—	37·4	37·4	37·6	37·3	37·4	37·4

There was no swelling of the subcutaneous tissue at the seat of injection, and no elevation of the temperature.

The horse was killed on the 16th August, and the *post-mortem* revealed the following :

Post-mortem Record.—The stomach, intestines, spleen, and mesenteric glands show no alteration. In the liver there are several pin-head, yellow, sharply defined tubercles, whose centre is calcified and whose periphery shows a stratified structure. Under the pleura of the right lung there are two yellow tubercles of the size of hemp seeds ; these are calcified at their centres, and they are surrounded by a delicate connective tissue capsule with a smooth internal surface. Under the pleura of the left lung there are three grey translucent tubercles, each of the size of a hemp seed ; these are sharply circumscribed and surrounded by sound lung tissue. The cervical organs show no alterations.

It will thus be seen that the grey translucent tubercles described by Nocard were present in the lungs of this horse. All three tubercles were dealt with in the way described in the beginning of this article, and then cut with the microtome and examined. They showed the structure of chronic inflammatory tubercles, and each contained in its centre a round worm. This horse, therefore, did not become affected with glanders after a single administration of a small quantity of glanders bacilli.

Another horse had for a long time a small quantity of glanders bacilli administered to him daily. The horse lived under conditions similar to those of sheep that daily visit a pasture infected with anthrax spores. Such sheep take in the spores of anthrax daily, but they first become affected when the conditions necessary for the germination of the spores they have taken in, that is to say, for infection, become developed. The same can also be observed in human beings who live in a locality in which cholera has broken out. In the intestine of such men cholera bacilli may be detected, but the infection first sets in when the necessary conditions arise in the intestine.

5. A mare, eighteen to twenty years old, received daily from the 13th to the 17th August the tenth part of a loopful of a pure culture of glanders bacilli obtained from the lymphatic glands of horse No 1 and grown on potato. The material was administered in the form of gelatine balls. The temperature of the animal was taken daily with the following result :

Day.	Morning.	Mid-day.	Evening.	
15th August	37·7	37·7	38·1	
16th ,,	37·5	37·7	37·8	
17th ,,	37·7	37·6	38·0	One ball given.
18th ,,	37·9	38·3	37·7	do.
19th ,,	37·5	37·6	38·0	do.
20th ,,	37·6	37·8	38·0	do.
21st ,,	37·7	37·9	38·2	do.
22nd ,,	38·2	38·4	38·2	do.
23rd ,,	37·3	38·0	38·3	do.
24th ,,	37·4	38·3	38·4	do.
25th ,,	38·3	38·2	38·8	do.
26th ,,	38·6	38·8	39·0	do.
27th ,,	38·6	38·9	39·3	do.
28th ,,	39·2	39·2	39·0	do.
29th ,,	38·8	39·2	39·4	do.
30th ,,	39·1	39·1	39·5	do.
31st ,,	39·3	39·3	39·4	
1st Sept.	38·7	38·8	39·3	
2nd ,,	39·3	38·7	39·4	
3rd ,,	39·1	39·4	39·7	
4th ,,	38·6	38·9	39·4	
5th ,,	39·1	39·1	39·0	
6th ,,	38·6	39·1	39·1	
7th ,,	38·7	39·1	39·0	
8th ,,	38·0	38·7	39·1	
9th ,,	38·0	38·3	38·4	
10th ,,	38·5	39·0	39·0	
11th ,,	38·4	38·8	38·5	
12th ,,	37·7	38·7	38·6	

Day.	Morning.	Mid-day.	Evening.	
13th Sept.	38·5	38·6	38·7	
14th ,,	38·6	38·9	38·9	
15th ,,	38·4	38·7	38·9	
16th ,,	38·4	38·6	38·7	
17th ,,	38·4	38·7	39·2	
18th ,,	38·0	38·0	38·5	
19th ,,	38·7	38·7	39·0	
20th ,,	38·5	39·2	39·3	
21st ,,	38·8	39·0	39·3	
22nd ,,	38·2	39·2	39·0	
23rd ,,	38·7	38·3	38·5	
24th ,,	37·8	38·1	38·4	
25th ,,	38·0	39·0	38·7	
26th ,,	38·1	39·2	38·7	
27th ,,	38·4	38·6	38·6	
28th ,,	38·3	38·9	38·6	
29th ,,	38·3	38·6	38·9	
30th ,,	38·2	38·7	38·3	
1st October	38·2	38·6	38·6	
2nd ,,	37·7	38·7	38·6	
3rd ,,	38·9	38·5	38·9	
4th ,,	38·1	38·0	38·4	
5th ,,	37·9	38·2	38·4	
6th ,,	37·9	38·3	38·4	

At midnight on the 6th October the horse was injected with ·5 ccm. of Preusse's mallein. The result of the test was as follows :—

Day.	Midnight.	6 a.m.	8 a.m.	10 a.m.	Noon.	2 p.m.	4 p.m.
6th October .	38·3	—	—	—	—	—	—
7th ,, .	—	37·9	38·0	38·1	38·3	38·0	38·4

It will be observed that no elevation of temperature occurred after the injection of the mallein.

The horse was killed on the 8th October and the *post-mortem* examination revealed the following :—

Post-mortem Record.—General condition bad. In the peritoneal cavity a litre of yellowish clear fluid. Peritoneum smooth. Mucous membrane of the stomach and intestines normal. On the outside of the pylorus, close to the head of the pancreas, there lies a walnut-sized nodule which on section proves to be a diseased lymphatic gland. In the interior of the nodule there are three pea-sized centres filled with a greyish-yellow degenerated material ; the other parts of the nodule are white and firm. A lymphatic gland of similar character and as large as a hazel nut is present on the outside of the duodenum. In the colic mesentery there is a packet of lymphatic glands as large as a hen's egg, the individual glands of which it is composed being as large as a hazel nut. In some of the glands there are numerous pin-head yellow centres of degeneration. Between the foramen dextrum and the muscular part of the diaphragm on the left side

there are five flat elevations in an area of the extent of the palm of the hand; these are 1 cm. thick, and their centres are composed of greyish-yellow, softened material.

In the liver, close under its capsule, there are two tubercles of the size of a hazel nut; the peripheral part of the tubercles is firm and greyish-white, while the central part is degenerated and yellow. Within the liver substance there are also present four greyish-white tubercles of the size of a hemp seed; the centre of these is opaque and the periphery reddened.

In the spleen there is a pea-sized tumour whose central part is softened while its peripheral part is composed of a firm white tissue.

In the lungs there are fourteen tubercles varying in size from a pin's head to a pea; the centres of these are greyish-yellow and opaque, while at the periphery they are greyish-white and surrounded by a red zone. The lungs also contain four translucent tubercles which are sharply defined and surrounded by healthy lung tissue.

In spite of the most careful examination no lesions were found in any of the other organs.

What was found in this case deserves special attention, inasmuch as two species of tubercles were present in the lungs, namely, the translucent grey tubercles surrounded by healthy lung tissue, and opaque grey tubercles, the neighbourhood of which was reddened. There were thus present in the lungs of this particular horse lesions similar to those described by Nocard as having been found in his experimental horses. What now did the microscopical examination of these tubercles show? The four translucent grey tubercles when imbedded and cut showed in their interior the well-known round worm, and the other tubercles, four of which were imbedded and cut in the same way, showed, in correspondence with their microscopic appearance, that they were glanders tubercles. The finer details of the structure of these tubercles is more minutely described on page 29.

Another point of interest is that the mucous membrane of the stomach and intestines showed no lesions, and that only a few of the lymphatic glands were affected, two of these being at the beginning and several at the end of the intestine, whereas all the other recognisable lymphatic glands in the mesentery and the whole of the chyle vessels were completely normal. It thus appears that the mucous membrane of the intestine is only slightly suitable for the settlement of glanders bacilli, but under certain favourable conditions not yet understood it possesses the property of allowing glanders bacilli which have reached the intestine to penetrate as far as the neighbouring lymphatic glands.

The result also teaches that the glanders tubercles in the lungs are secondary in their nature, and referable to the glanderous lesions in the before-mentioned lymphatic glands.

Lastly, it remains to be mentioned that an elevation of temperature was not observed in the course of the second day as in horses Nos. 1 to 3, but first on the fifth day after the beginning of the administration of glanders bacilli.

Part of the degenerated products from the diseased lymphatic glands was sown out on glycerine agar. After three days numerous

colonies of glanders bacilli had developed. Glanders bacilli were detected in cover-glass preparations from the degenerated products, and three guinea-pigs which had been inoculated with part of the diseased lymphatic glands contracted glanders; from the glandered testicles of these guinea-pigs pure cultures of glanders bacilli were again obtained.

The result of the experiments described in the preceding pages shows that the method pointed out by Nocard is not calculated to give rise to primary glanders of the lung of the horse. On the contrary, they show that when horses become affected through the stomach and intestines after taking in glanders bacilli, the glanders lesions that develop in the lungs are of a secondary nature. Hence, the opinion which I pronounced many years ago that primary glanders of the lung, if it occurs at all, is extremely rare, has not yet been controverted. I experience a sort of satisfaction that Johne has now adopted this view, and in the interest of the matter I refrain from all discussion which does not touch the kernel of this point. I would like, however, to say that in spite of a most extensive experience I have not yet observed a case of primary glanders of the lung of the horse. If one regards the "grey translucent" tubercles in the lungs as lesions of glanders one can arrive at such a conclusion, but how erroneous this view is has been shown by the careful and diligent examination of such tubercles in the Pathological Institute. Hundreds of such tubercles have been examined, and in the whole of them the often mentioned round worm has been detected. Consequently, two possibilities remain; either Nocard has made a mistake, or French horses behave differently from others with regard to glanders bacilli. The latter possibility is pretty well excluded, and there therefore remains, when the experiments carried out in the Pathological Institute are taken into consideration, only the first possibility. In such an important question as this I must be candid, and I therefore ask Professor Nocard's pardon for being so frank. In this sense I also assert that Nocard's experiments do not at all show that the grey translucent tubercles contain glanders bacilli. Nocard, in the case of a horse that was fed with glanders bacilli and afterwards became glandered, had the animal killed thirteen days after feeding with glanders bacilli, then cut out of the lungs a large number of grey translucent tubercles, rubbed these up, and from them inoculated potatoes, glycerine agar, and guinea-pigs. If now after inoculations from the rubbed-up material some few colonies of glanders bacilli had grown on potato or glycerine agar, or one or other of the inoculated guinea-pigs had contracted glanders after inoculation, this result would, nevertheless, not show that the tubercles were glanderous in their nature, and, especially, they would not show that these tubercles owed their origin to the struggle between the tissues and the glanders bacilli. Just as well justified is the conclusion that the tubercles were of a simple inflammatory nature, and had first become the carriers of glanders bacilli owing to their having been accidentally situated in a glandered horse and thus in a quite accidental manner become contaminated with glanders bacilli. I will observe further that I have succeeded in detecting glanders bacilli in the blood of horses in cases of acute glanders, and that from such

blood I was able to cultivate glanders bacilli in a state of purity on potatoes and glycerine agar, or to infect guinea-pigs. Furthermore, I might mention that Nocard has expressed different views regarding the development of the grey translucent tubercles. Whereas he formerly described these as the result of a healing process, and compared them with cicatrices, he now asserts that the grey translucent tubercles represent the young forms of glanderous lesions of the lungs. He now compares them with a granulation which at a later stage becomes fibrous, that is to say, cicatrises. Consequently, there is also another important question to decide, namely, whether there occur in the lungs of horses young glanders tubercles which in their anatomical structure can be compared with a granulation. I give hereafter the results of the investigations carried out in the Pathological Institute, but I will preface them by saying that I have nothing new to report, and that the facts observed are in agreement with those already published.[1]

The metastatic glanders tubercle in the lungs of the horse takes the form of a small pneumonic area ; it is reddened and not sharply defined, and passes gradually into the neighbouring sound lung tissue. In quite a short time the central part of this hepatisation tubercle becomes opaque. Even at this time the tubercle is distinguished by characteristic features. The central opaque grey part has not the form of a smooth surfaced sphere, but is beset with small projections ; sometimes it is wedge shaped. The central part of the tubercle is always sharply defined as compared with the redder peripheral part of the same. The freshly exposed cut surface of a tubercle appears granular like pieces of lung which are the seat of fibrinous inflammation, and this is true both of the central and the peripheral part of the tubercle. The cut surface of quite recent glanders tubercles is thus somewhat moist, grey, and opaque in its centre, but reddened at its periphery. Older glanders tubercles are on section no longer granular ; this is especially true of the centre of the tubercle, which becomes smooth, dry, and greyish-yellow, while around it there forms a thin zone of granulation tissue, the colour of which varies between red and greyish-white. The number of connective-tissue elements in this zone gradually increases, until ultimately the opaque, dry, greyish-yellow central part is surrounded by a translucent grey capsule. In the outward direction this capsule passes gradually into the neighbouring healthy lung tissue ; only occasionally is this transition effected by means of a red zone.

Inasmuch as I have within recent years, as already mentioned, again had the opportunity to examine the lungs of many glandered horses, I will observe that along with older tubercles there were always to be recognised younger ones showing all stages of the before-described inflammatory process.

With regard to the histological character of glanders tubercles the following points may be mentioned. The redness of young tubercles is caused by marked fulness of the capillary vessels. The loops of the same are so wide that the partition walls of the alveoli appear broader and the alveoli themselves smaller. In addition the alveoli in young glanders tubercles are filled with fluid and with red blood corpuscles ; the latter, however, soon degenerate, and their colouring

[1] Archiv für wissen. und prakt. Thierheil, vol. xxi., p. 376.

matter appears in the form of very fine granules in the fluid. Frequently numerous alveoli, the infundibula belonging to them, and the neighbouring minute bronchi are closely packed with finely divided blood pigment. At the same time swollen detached epithelial cells and round cells appear in the fluid exudate. The number of these increases more and more until they form the bulk of the alveolar contents. The round cells resemble lymphoid cells; the nucleus of the same is uniformly and deeply stained by hæmatoxylin and the other stains in common use. Between the lymphoid cells their lie multinuclear leucocytes. The contents of the alveoli then coagulate and fibrin threads are formed. It is the formation of fibrin that causes the cut surface of a young glanders tubercle to have a granular appearance. The young glanders tubercle is thus the product of a fibrinous miliary pneumonia, and it is therefore not a granulation as Nocard asserts, but a tubercle of hepatisation.

Retrogressive changes now set in. In the centre of the same the alveolar septa, the capillary vessels, and the contents of the alveoli die, and therewith begins the decolorisation. The centre becomes grey and opaque, and on section it now appears smooth.

In the dissolution of the nuclei of the round cells the following process may be recognised. The chromatin of the cell nucleus does not become destroyed as in other processes of mortification, but remains even when the nuclei are completely degenerated. In the first place the nucleus falls into many small droplets which lie close together and form a small heap. The latter still shows roughly the form of the nucleus. At a later stage some of the granules of the nuclear material become isolated and distributed in the plasma body of the cell, or the nuclear mass disintegrates and becomes uniformly distributed in the cell substance. During this the chromatin has not changed, and on treatment with staining reagents it even now shows all the properties of the nuclear material. Of many cells that have perished only the chromatic substance of the nucleus is retained, in the form of globules or droplets, which are not infrequently connected with one another by fine strings. The droplets are often beset with branched processes to the ends of which irregular branched remains of the plasma body of the cell are attached. At the same time the remains of the cell body lying between the chromatin material, or the fat derived from the same, gradually diminishes, until ultimately the chromatin masses (the fragments of the nucleus) lie close together. A peculiar detritus develops in the centre of the glanders tubercle; this material cannot be compared either with pus or cheese. On account of its richness in chromatin it stains very deeply when sections are laid in the stains in common use. The richness in chromatin is also the reason why the dead part of old glanders tubercles is tough and somewhat dry. On the other hand—and I have already in former years called attention to this point—calcification of the dead part of a glanders tubercle could never be recognised, although several hundreds of old glanders tubercles from the most varied series of glandered horses have been cut with the microtome and afterwards minutely examined. I will add further that not once could a trace of lime be found in old glanders tubercles, not even in the examination of quite old tubercles in which the still persisting chromatin mass was surrounded by a thick connective tissue capsule. This would appear

to indicate, as is borne out also by the persistence of the chromatin, that the metabolic products of glanders bacilli, namely, mallein, act like an acid, and therefore prevent the precipitation of lime salts. When out of the great number of lungs of glandered horses examined lime could be found in one tubercle, that proved on microscopic examination to be a non-glandered tubercle. Furthermore, chromatic substance could not be detected in the dead parts of the entozoic tubercles of the lung of the horse, and the occurrence of chromatin is thus, according to our present knowledge, by itself a certain mark by which one may prove the glanderous nature of a tubercle in the lungs.

This remarkable change of the cell nucleus in glandered processes, which may be reckoned as an example of karyorrhexis[1] or karyochisis (Klebs), was first observed by Unna,[2] and designated chromatotexis or liquefaction of the nucleus. Regarding the process Unna remarks as follows: " Inasmuch as this peculiar degeneration of the nucleus plays an important rôle in the mature glanders tubercle, and does not appear to have been hitherto sufficiently well distinguished from other degenerations of the nucleus, I will name it nuclear liquefaction (chromatotexis). It is not pathognomonic for glanders, but is found also in other necrotic processes, but at the same time not in such a pronounced degree and so exclusively as here." " The fate of the nuclein in the glanders tubercle is of great interest." " For it exists here not simply in the form of granules and drops, but especially in the shape of repeatedly twisted and curved threads which are always at one or both ends attached to chromatin drops."

Ehrich[3] also has also given accurate accounts of the process designated nuclear liquefaction.

Around the destroyed central part of the glanders tubercle which is rich in chromatin there forms in the meantime a connective tissue capsule, which, in the outward direction, passes gradually into the normal lung tissue. The central part of the tubercle is composed of the above described detritus rich in chromatin, and the capsule is distinguished by the presence of wavy connective tissue strands which are very similar to those of fibrous connective tissue. In the numerous crevices and spaces between the strands there lie round cells with nuclei which are easily stained. In passing towards the periphery of the tubercle one finds all transitions between these spaces and the pulmonary alveoli. Many of the latter are filled with cellular exudate, others are pressed together to form small longitudinal clefts. The partition walls of the alveoli are very broad, and the tissue of which they are composed has taken on a connective-tissue character. This stratum thus represents a transitional layer between the tubercle and the neighbouring normal lung tissue, and there is therefore no sharp boundary between the two. The capsule of the glanders tubercle is also a product of the chronic process of irritation.

In very old glanders tubercles the boundary between the central dead part of the tubercle and its connective tissue capsule is very sharp, and it is then not possible to make out that the glanders

[1] Schmaus und Albrecht, Ueber Karyorrhexis. Archiv für pathologische Anatomie. Bd. 138.
[2] Unna, Die Histopathologie der Hautkrankheiten. 1894.
[3] Erich, Zur Symptomatologie und Pathologie des Rotzes beim Menschen. Beiträge zur klinischen Chirurgie. XVII. S. 1.

tubercle is the product of a miliary pneumonia. At this boundary
there takes place a slow degeneration of the dead inflamed lung
tissue, and here, also nuclear liquefaction is observable. After this
the degeneration reaches as far as the capsule, the inner face of which
becomes irritated by the detritus, and this slight irritation is the reason
why giant cells and plasma cells form on this surface of the capsule.
Both these kinds of cell are distinguished by their uncommon size.
Sometimes the giant cells and the plasma cells occur in large numbers
on the inner face of the capsule, and they are found also here and there
in the granulation tissue or connective tissue which forms the capsule.
On the other hand neither giant cells nor plasma cells can be recog-
nised in fresh glanders tubercles. This must be the reason why the
occurrence of giant cells in glanders tubercles has in the literature of the
subject not been mentioned or has even been denied.[1] At a still later
stage the giant cells also perish, and here again it may be observed that
during the destruction of the cell nuclei the chromatin is preserved.
Many giant cells of old glanders tubercles frequently contain among
their numerous nuclei some whose chromatin is liquefied and distri-
buted in the form of granules or small round particles in the plasma
body. The granules have a variable size, but most of them are
about the size of cocci. In other cells in which the liquefaction of
the nucleus has begun the fragments of the nucleus lie together in
little heaps. This liquefaction of the nucleus does not occur in the
giant cells of tuberculosis, or in those which are sometimes found in
the immediate neighbourhood of the entozoic tubercles, and by this
character these cells may be distinguished from those of the glanders
tubercle. Whereas in the giant cells of tuberculosis the cell nuclei
are still recognisable long after the chromatin has disappeared, the
latter can still be recognised in the glanders tubercle even when the
nucleus and the plasma of the cell have perished. Most of the giant cells
of the glanders tubercle reach by means of branched processes into the
detritus mass resulting from the degeneration of the cells, whereas the
nuclei appear to escape the degeneration, and are thus to be found on
the side of the cell turned toward the capsule.

The results of the preceding experiments I would sum up as
follows :—

1. Primary glanders of the lung is not the result of an infection
proceeding outwards from the digestive apparatus.

2. The occurrence of primary glanders of the lung has certainly not
yet been demonstrated.

3. The grey translucent tubercles in the lung of the horse are not
glanderous, but of a simple inflammatory nature and determined by
a parasite which has also been detected in the kidneys of the horse.

4. The glanders tubercle in the lung of the horse is a small hepati-
sation tubercle which degenerates in a peculiar way (chromatotexis).

5. Old glanders tubercles contain giant cells.

6. The glanders tubercles in the lungs do not calcify, but the entozoic
tubercles do.

In conclusion, still another word. In the text-book of Pathological
Anatomy by Birch-Hirschfeld, fifth edition, page 361, Herr Johne has

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[1] [This is an error on the part of Professor Schütz. The occurrence of giant cells in glanders
tubercles was pointed out in an article entitled "The Pulmonary Lesions of Glanders," which
appeared in Vol. VIII. of this Journal, pp. 55 and 56.—J. M'F.]

criticised the instruction regarding the anatomical characters of glan-
derous lesions in the lungs of the horse which was drawn up at the
instance of the Technical Deputation for veterinary affairs, as if this
instruction had been a scientific work of the first rank. He has, thereby,
in my opinion, done the instruction too much honour. An instruction
can only contain the rule for a technical opinion, and nothing more ought
to be expected. At the same time the above-reported results of the
tedious investigations carried out in the Pathological Institute show
how correct was the opinion pronounced in the instruction.

NOTES ON THE THERAPEUTIC AND TOXICO-
LOGICAL EFFECTS OF CHINOSOL.

By F. HOBDAY, F.R.C.V.S., Professor of Therapeutics, Royal
Veterinary College, London.

CHINOSOL, a comparatively new drug, appears to be making its way
to the front, both in human and veterinary medical practice, and, on
that account, doubtless, the following remarks upon some of its thera-
peutic and toxicological properties may prove of value.

It occurs as a light yellowish powder, having a curious, not un-
pleasant, odour; and it is conveniently prepared in 8 and 15 gr.
tablets.

Chinosol is stated to be a pure chemical compound belonging to
the Quinoline group, to be readily soluble in water, and to have for
its formula $C_9H_6NKSO_4$. Its properties are represented as antiseptic,
disinfectant, and deodorant. It is also stated on the label and in
the pamphlets sent out to be "absolutely non-poisonous and non-
irritant," although, with regard to the latter, it is said that "when
pure chinosol is applied to open wounds it causes a transient
burning sensation." Dr Klein, reporting upon its bacteriological
properties, and comparing its effects with those of phenol, states
that "chinosol kills staphylococcus pyogenes aureus in five minutes
in a solution of 1 in 150, whereas it takes a 5 per cent. solution
of absolute phenol to produce the same result. A chinosol solution
of 1 in 100 kills the spores of anthrax in five minutes, whereas a
6 per cent. solution of absolute phenol does not in the least interfere
with their vitality even after forty-eight hours contact." Bonnema has
also investigated its action upon bacteria, and he finds that a 5 per
cent. solution will arrest the growth of staphylococcus. It is found to
hinder decomposition, to check alcoholic and lactic fermentation, and
to retard the coagulation of albumen.[1]

Mr Edwards, M.R.C.V.S., has contributed to the *Veterinary Record*[2]
a short article giving the result of his experience of its internal use in
15 grain doses every six hours for three consecutive days, in a cow
suffering from tympanites; also of the external use of a solution of
chinosol in two wound cases; whilst in the same year[3] there appeared
amongst the extracts from foreign Journals some remarks by Dr

[1] "The Year Book of Treatment" for 1898, page 456.
[2] Veterinary Record, 6th March 1897.
[3] Idem, 14th August 1897.

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Zacher, stating that chinosol solution (1 in 1000) is a good substitute for sublimate solution, possessing the advantages of being comparatively non-poisonous, and, in concentrated state, not at all caustic.

In the *Lancet*[1] there is a summary of the experience of Dr Cipriani with chinosol in the treatment of tuberculosis in the human subject, a rapid and apparently permanent improvement following its use in eight cases. It was either administered by the mouth or injected locally.

During the past nine months we have used the drug extensively in both the canine and equine clinique, paying particular attention to the properties which it is stated to possess. The variety used throughout was that which is usually sent out in bottles, unless where otherwise stated.

First with regard to its antiseptic properties. Wounds of all kinds were treated with solutions of from 1 in 60 to 1 in 1200 with the most satisfactory effect, the strength which we found to give the best results being from half a grain to a grain to the ounce. Upon fœtid ulcerating wounds a proportion of 1 in 480 speedily caused a healthy appearance and entire absence of pus, the application being made once or twice daily. In several cases we were able to compare the effects of solutions of this strength (in treating wounds on the same animal) with solutions of lysol and creolin, the chinosol giving decidedly the best result. In the form of powder, as a dry dressing, when mixed with boracic acid, zinc oxide, or starch, and compared with iodoform used similarly, the sequelæ appeared about the same. When the pure powder was applied to wounds the effect was to cause a good deal of pain, the animals showing signs of great irritation for two or three minutes, and the raw surface turning a blackish-brown colour. In one case, in which a fresh skin wound was treated by the parts being carefully dusted over with 6 grains of pure chinosol and the edges drawn together with aseptic silk, the wounded area kept very tender, and on the third day was perceptibly swollen. On the fourth and fifth days this increased very much and fluctuated on pressure; on the sixth day the lancet was applied and gave exit to a quantity of thick, dark brown, syrupy fluid.

As a disinfectant to the hands, skin, and suture threads it was employed in solutions of from 1 to 1000 to 1 in 60 without any signs of irritation either to the hands of the operator or the skin of the patient. With instruments, however, care must be taken, and the solution used should be carefully measured. On several occasions, when we neglected to do this, the instruments lost their edge, the steel parts became coated with greenish-black spots which were very troublesome to entirely remove, and in those which had white bone handles the latter became discoloured and rough to the touch. The solution recommended for this purpose consists of 1 in 1200, and, if the instruments are to remain in it for anything like an hour, this strength certainly should not be exceeded. In order to watch the effect of a stronger preparation on instruments, a white-handled scalpel was placed in a solution made by dissolving a 15 grain tablet in 8 ounces of water. In a short time there was a distinct zone of discoloration around the steel part, and the knife was becoming marked with greenish-black areas; in an hour these areas

[1] Lancet, 27th November 1897.

became almost black and the bone handle felt rough to the touch. It was not removed until eighteen hours later, when both the handle and steel were very much stained, and evidences of its effect are still present although this occurred nearly five months ago.

As a deodoriser for the hands or for fœtid wounds, solutions of the same strength as those used for disinfectant purposes acted satisfactorily.

The following cases will illustrate the amount and strength of the applications used externally, principally in the treatment of mange.

CASE 1.—Wire haired terrier, 14 lbs. weight, having a fair length of coat, and suffering from follicular mange. Well applied with friction all over the body, for ten minutes, a solution of 1 ounce of crude chinosol (powder F.) in 10 ounces of water. This caused slight irritation, rigors, and frothing at the mouth ; these symptoms soon passed off and no bad result followed. The next day an ointment consisting of 2 ounces of crude chinosol (powder F.) in 3 ounces of vaseline was well applied all over the body. No noticeable effect followed.

CASE 2.—Bull dog, about 30 lbs. weight, suffering from follicular mange. This dog was dressed all over the body every third day for twelve days, in the month of October, with an ointment composed of 1 drachm of chinosol to 4 ounces of lard, one fourth part being used at a time.

During the first fortnight of November the patient was similarly treated with ointment, in the proportion of 10 grains to the ounce. During December and January the ointment was applied once a week. The dog improved in condition and the hair that was already there grew longer, but fresh hair did not come through the diseased patches, although the mange did not spread.

CASE 3.—Bull dog, ten months old, about 16 lbs. weight, suffering from follicular mange. The body was thoroughly well dressed with a lotion composed of 1 drachm of chinosol dissolved in 40 ounces of water, the application being made with friction for eight minutes. There was slight irritation for about four or five minutes, but after that nothing else was noteworthy, the animal feeding as usual.

Three days later a lotion of half a drachm of chinosol dissolved in 13 ounces of water was similarly applied, and two days after that another dressing in the proportion of half a drachm to 10 ounces. In each case slight irritation was observed for a few minutes after the dressings were applied, but no dulness or other bad symptom. Seven days later the animal died without any appreciable reason to account for it, and unfortunately no *post-mortem* examination was made.

CASE 4.—Irish terrier, four years, about 24 lbs., suffering from sarcoptic mange. This animal was well dressed all over the body, after the hair had been removed, with an ointment composed of chinosol ʒij., sulphur sub. ʒiv., vaselin ℥iv. After the application the animal was continually sneezing for about twenty minutes, and shaking its body at short intervals. The ointment was washed off ten days later, no bad symptom ensuing.

CASE 5.—Airedale dog, about 35 lbs., suffering from sarcoptic mange. This animal was dressed in a similar manner to Case 3, there being nothing worthy of remark.

CASE 6.—Toy terrier mongrel, 5 lbs. weight, about three months old, suffering from mange.

12.55 P.M.   Thoroughly applied for seven minutes all over the body a solution of 4 drachms of chinosol in 4 ounces of water.

1.10.   Temperature 99°.   Showed signs of irritation, salivating very freely.

1.45 and 2.15.   Temperature 102·2°.   Seemed uncomfortable.

After this nothing abnormal was noticeable, the animal eating its food as usual.

CASE 7.—Fox terrier, about two years old, about 15 lbs. weight, very poor in condition, suffering from follicular mange.

15th February.   Well applied for five minutes all over the body an ointment composed of 1 drachm of chinosol to 2 ounces of vaseline. The hair of the coat had previously been clipped close.   Nothing noteworthy resulted, and the dressing was washed off ten days later.

11th March.   The whole body was thoroughly dressed with a solution of 1 drachm chinosol in 6 ounces of water.

12th March.   Feeding well.   Dressed with a solution of 1 drachm of chinosol in 4 ounces of water.

14th March.   Dressed with solution of 1 drachm of chinosol, 4 ounces of glycerine, and 6 ounces of water.   The animal did not show any signs of dulness or irritation, and ate heartily.

15th March.   Applied 1 drachm chinosol, and 2 ounces each of glycerine and water.   This was washed off on the 19th, the skin not being at all tender.

CASE 8.—Bull pup, 14 lbs. weight, suffering very severely from follicular mange.   Well applied for ten minutes an ointment composed of 1 drachm of chinosol, half a drachm of olive oil, and 2 ounces of vaseline.   No signs of irritation followed the application.

CASE 9.—Cat, 5½ lbs. weight, three or four years old, suffering from sarcoptic mange.

8th October.   3.50 P.M.   Well applied with friction for five minutes all over the body a solution of 5½ drachms of crude chinosol (powder F.) dissolved in 6 ounces of water.

4.15.   Frothing slightly at the mouth.

4.30.   Ate food greedily.   A second dressing consisting of 10 grains of chinosol, 10 minims of olive oil, and 6 drachms of lard was applied on the 15th to the head and neck, without any bad effect.

CASE 10.—Cat, 5 lbs. weight, suffering from fleas.

1.25 P.M.   Well applied for five minutes a solution of 1 drachm of chinosol in 8 ounces of water.

1.30.   Frothing at the mouth, occasionally attempting to lick the skin, the taste evidently being very unpleasant.

3.30.   Seemed dazed and weak ; staggered when walking ; salivating freely.

4.30.   As at 3.30 ; mewed when touched, body surface cold.   The next morning the animal was found dead.

CASE 11.—Cat, 6 lbs weight, suffering from sarcoptic mange.   Well applied with friction all over the body for five minutes 1 drachm of chinosol mixed up with 1 ounce of lard.   No bad effect followed.

CASE 12.—Cat, about three years old, 7 lbs. weight, suffering from sarcoptic mange.

17th February. 12.30 P.M. After clipping the hair off, a solution of 3 drachms of chinosol in 6 ounces of water was thoroughly applied all over the body for ten minutes. The animal appeared dull afterwards and refused to feed.

18th February. Dull; not feeding or drinking at all; mewing continuously, weak in the hind quarters; staggered when walking.

19th February. As on 18th. Temperature too low for the thermometer to register.

20th February. As on 19th, but completely prostrate and unable to rise. Died during the night.

Post-mortem revealed all the organs of the body to be normal, and no smell of chinosol in any of them. The smell on the skin was very distinct.

Being desirous of finding the dose of chinosol which could be used in veterinary patients subcutaneously for the treatment of tuberculosis, and also of setting at rest the question as to whether the deaths of Cases 3, 10, and 12 could be attributed to any toxic or irritant property of the drug, the following observations were made:

CASE 13.—A cat, 6 months old, weighing 5½ lbs. received 1½ drachms of chinosol dissolved in half an ounce of warm water at 1.30 P.M. At 3.0 P.M. the animal was prostrate, the pupils being widely dilated, and the temperature too low to be registered on the thermometer. This condition was maintained until death occurred about 5 o'clock.

Post-mortem made eighteen hours afterwards revealed the presence of frothy spume in the nostrils and a lot of frothy saliva in the mouth. The œsophagus and stomach contained a thin frothy fluid. All the other organs appeared normal; the brain was not examined.

Being now satisfied from the experience of Case 1 that chinosol possessed toxic properties, the following observations were made with a view to finding out the amount which might be allowed to be absorbed into the system with safety.

CASE 14.—Cat, weighing 5½ lbs. received 1 grain dissolved in a drachm of water subcutaneously. No noticeable effect was observed within the next twenty-four hours, after which the animal was destroyed by a dose of hydrocyanic acid. On *post-mortem* examination no smell or trace of the chinosol could be found at the seat of injection.

CASE 15.—Cat, about eight months old, 4½ lbs. weight.

1.22 P.M. Temperature 102.6°. Injected subcutaneously 10 grains of chinosol dissolved in a drachm of water.

1.30. Very quiet; no signs of irritation.

1.50. Salivating very freely; the saliva being ropy and hanging from the mouth and around the lips.

2.0. Salivating as at 1.50; respirations increased; unsteady movements when walking; temperature 100·2°; very quiet.

2.10. Prostrate; could not rise without assistance, or walk even a yard without falling over on the side; gasping; respirations irregular; no convulsions.

2.15. As at 2.10.

2.30. Weaker; temperature 98·1°; pulse 186 per minute; body surface cold; quite a pool of saliva on the floor.

3.15. Completely prostrate; still salivating freely; temperature too low to register on the thermometer (below 95°).

3.35.   Pulse 160.

3.50.   Seemed brighter.

5.15.   Spasmodic gasps at varying intervals, the head being slightly drawn towards the chest ; the inspiratory effort distinctly longer than the expiratory ; heart beating irregularly and very feebly.

5.30.   Respirations ceased ; the heart beating feebly but perceptibly for three minutes longer.

Post-mortem examination made the next day revealed a lot of frothy saliva in the mouth, pharynx, œsophagus, and stomach, the other organs being normal.   At the seat of injection there was an area showing distinctly the yellow colour of chinosol and possessing its characteristic odour.

CASE 16.—Cat, 4½ lbs., very thin, and suspicious of tuberculosis.

9th March.   Injected subcutaneously half a grain dissolved in 30 minims of water.   The animal fed well, and, beyond slight dulness, nothing was noticeable.

11th March.   1.0 P.M.   Temperature 102·4°.   Injected 1 grain dissolved in 30 minims of water.

1.30.   Temperature 100·4°.   Stringy, sticky saliva hanging from the mouth ; constant continuous cough as if spasm of the larynx.

1.50.   Less saliva ; animal brighter.

3.30 ; 5.0 ; 6.30 ; 10.0 P.M.   Nothing noticeable ; fed and walked well.

12th March.   Injected subcutaneously half a grain dissolved in 30 minims of water.

14th March.   Subcutaneous injection of one quarter of a grain dissolved in half a drachm of water.   At the usual seats of injection there was a decided, tender swelling.

15th March.   This swelling was less in size.   Injected half a grain dissolved in 1 drachm of water.   Animal bright and feeding well.

16th to 19th.   Injected daily half a grain dissolved in 1 drachm of water.   Appetite good.

21st and 22nd.   As on the 19th.   Swelling at seat of injection of the 1 in 60 solution on the 12th still perceptible but not tender, and much less in size.   No swelling at seat of the 1 in 120 injections. Animal bright and lively.

CASE 17.—Cat, 3 lbs. weight, about three months old.

3.45 P.M.   Temperature 103°.   Subcutaneously injected 5 grains of chinosol, dissolved in 1½ drachms of water.

3.50.   Staggering gait ; quantity of stringy saliva around the jaws.

3.55.   Temperature 101·5°.   Otherwise as at 3.50.

4.0.   Prostrate ; could not rise ; quite a pool of saliva around the lips ; pupils about two-thirds dilated.

4.7.   Mewed a few times at intervals.

4.20.   Temperature 100°.   Pupils widely dilated ; occasional convulsive spasms of the muscles of the legs ; still mewing occasionally.

4.30.   Temperature 99°.   Quiet ; apparently unconscious ; otherwise as at 4.20.

4.55.   Temperature too low to register ; still salivating freely ; heart beating very irregularly.

5.5.   Pulse scarcely perceptible.

Died a short time after.

Post-mortem about eighteen hours later, revealed a distinct yellow colour and odour of chinosol at the seat of injection. Nothing else noticeable. There was no froth in the pharynx, œsophagus, or stomach.

CASE 18.—Cat, weight 5 lbs. 6 oz.

1.0 P.M. Temperature 101·6°. Subcutaneously injected 2 grains, dissolved in 1 drachm of water.

1.30. Temperature 99·6°. Immense quantity of stringy saliva hanging from both sides of the mouth; continual sneezing, and a constant short sharp cough as if suffering from spasm of the larynx. Breathing through the mouth.

1.50. Symptoms apparently passing off.

3.30; 5.0; 6.30. Dull; not salivating.

The next day no ill effect was observable, and 1 grain dissolved in half a drachm of water was injected subcutaneously. This caused slight salivation, but no other symptom worthy of remark. The animal was destroyed by hydrocyanic acid six hours later, a *post-mortem* examination revealing an area of blood extravasation at the seat of inoculation. The internal organs appeared normal.

CASE 19.—Cat, 5 lbs. weight, very thin and emaciated, suffering from tuberculosis, the enlarged mesenteric glands being able to be felt through the abdominal walls.

4.30 P.M. One grain of chinosol dissolved in half a drachm of water was injected subcutaneously.

4.40. Gasping; thick ropy saliva coming from the mouth.

5.40. Prostrate; unable to rise; great salivation; frothy spume coming from the nostrils at each expiration. Respirations 48, snatchy and gasping; pulse 108; temperature below 95°; body surface cold; semi-comatose; pupils about two-thirds dilated.

Death occurred during the evening.

Post-mortem revealed the lungs and intestinal lymphatic glands to be badly affected with tuberculosis. The epiglottis had some frothy mucus on it with a yellowish tinge and odour of chinosol. The stomach contained frothy mucus with a faint odour of chinosol. The pelvis of the right kidney was full of watery fluid, having a faint but distinct odour of chinosol. These organs were all carefully cut with a clean fresh knife, which had not been in contact with the subcutaneous seat of inoculation. The latter place was slightly tinged yellow and had a distinct odour of chinosol.

CASE 20.—Irish terrier, about 16 lbs. weight.

12.30 P.M. Subcutaneous injection of 10 grains dissolved in a drachm of water.

1.30. Thick ropy saliva hanging from the mouth.

2.30. No salivation; appeared all right.

Nothing further was observed, the animal eating his food heartily.

CASE 21.—Fox terrier, very fat, weighing 28 lbs.

4.30 P.M. Subcutaneous injection of 18 grains of chinosol dissolved in an ounce of water.

4.45. Slight champing of the jaws, otherwise nothing noticeable.

Nothing further was observed.

CASE 22.—Toy mongrel, weight 5 lbs.

12.50 P.M. Temperature 101·2°. Subcutaneously 10 grains of chinosol in a drachm of water.

1.20.   Temperature 100·8°.   Salivating very freely.

3.30.   Dull.

The animal was destroyed twenty-four hours later, nothing being noticeable at the seat of injection.

CASE 23.—In a dog weighing 6 lbs., a subcutaneous injection of 6 grains dissolved in half a drachm of water caused no noteworthy symptom beyond signs of irritation for a few minutes after the injection.   Five days later 10 grains dissolved in 1 drachm of water caused local irritation at the point of insertion, but nothing else.   A month later 20 grains in 1 drachm of water caused death in about four hours, the symptoms shown being a stilty straddling gait, continual jerky cough, and profuse flow of stringy saliva, and the temperature too low to be taken.

Post-mortem revealed the presence of a small amount of fluid around the epiglottis, and the heart cavities almost empty.   The bladder was full, and I fancied that the urine had a slight odour of chinosol.

CASE 24.—A fox terrier received subcutaneously 1 drachm of a 1-200 solution, and 1 drachm of a 1-600 solution.   Both became absorbed without producing any signs of irritation, beyond a little at the time of injection.

With a view to testing the dosage and so-called non-irritant properties of chinosol on some of the larger animals, the following observations were made upon two donkeys :—

CASE 25 received 2½ drachms of a 1 to 4 solution of chinosol in water subcutaneously injected in the region of the neck, and a like amount in the axilla.   These caused uneasiness and irritation for about five minutes, and on the next and twelve following days there was a tender swelling in each place.

CASE 26 received similar injections with a like result.   A solution of 1 in 600 was then tried, but even here a sore swelling was produced lasting for two days.

The value of the drug as a vermifuge for the dog was tried in a few cases, but without success, besides which these animals object very much to its unpleasant taste, frothing very much at the mouth after its administration.   One drachm dissolved in 6 ounces of water, when given to a donkey, caused sneezing and coughing for four minutes almost continuously ; whilst afterwards for four hours the animal had a dry wheezing cough at intervals at first of about twenty seconds, the intervals gradually getting longer.

Summarising the results, it may be said :—

1. That Chinosol acts well as an antiseptic, disinfectant, and deodorant when used in certain proportions.

2. That its action is better marked when used as a lotion than when used as a powder.

3. That the powder is not suitable for use on fresh wounds unless diluted in some way or other.

4. That for the disinfection of instruments care must be taken not to make the solution too concentrated.

5. That the drug possesses toxic properties.

6. That if used subcutaneously in too concentrated a form it will produce local irritation and swelling. The strength recommended for subcutaneous injection in human practice is from 1 in 600 to 1 in 200, and certainly in veterinary patients this should not be exceeded.

7. That the cat is very susceptible to its action, and that in this animal much more care is necessary to guard against toxic symptoms than in the case of the dog. In the cat, if subcutaneously injected, the extreme limit of dose should be one-sixteenth of a grain for each lb. body weight, and in the dog one-eighth of a grain per lb.

8. That chinosol is not rapidly absorbed from the unbroken skin of the dog, and can be applied for several days in succession even in fairly concentrated solutions to the skin of this animal without producing eruptions or sores.

9. That the chief symptoms of poisoning are: Sneezing and coughing, an increased flow of thick ropy saliva; subnormal temperature; staggering gait, commencing with loss of motor power in the hind quarters; great prostration, and ultimately death from failure of the heart's action.

10. That the chief *post-mortem* characteristic is the smell of chinosol on or in some part of the body; whilst another symptom to be looked for is the presence of frothy saliva in the pharynx, œsophagus, or stomach.

INFECTIVE VENEREAL TUMOURS IN DOGS.[1]

By G. BELLINGHAM SMITH, M.B., B.S., and J. W. WASHBOURN, M.D.

SINCE the beginning of the year 1896 we have had under observation a series of contagious tumours on the genital organs of dogs. The contagium is conveyed in the act of coitus, and the tumours are in this respect comparable to the venereal tumours met with in man. This series seems to us worthy of putting on record, not only from the light it throws upon the ætiology of venereal tumours, but also as a slight contribution to the general question of the infective nature of tumours.

History of Infection.—From January to June 1894, a dog, A, served twelve bitches, eleven of whom became infected. About a month after pupping there was noticed in each case a growth in the vaginal wall, somewhat resembling a raspberry. The growth gradually increased in size and extent, until in some cases the whole of the vagina was filled with a mass as large as an orange.

An examination of the dog A revealed the presence of a similar growth, situated on the penis behind the corona.

The bitches were of various ages, the younger being less severely affected than the older. The oldest were very severely affected, and had to be killed.

Constant bleeding from the vagina was in every case the symptom which led to the detection of the growth. In none of the cases was there any antecedent purulent discharge.

Every bitch affected was operated upon by veterinary surgeons for the removal of the growths. Four (Nos. 1, 2, 3, and 4) were temporarily cured, but the growth reappeared after pupping. Further

treatment in these cases has resulted in a permanent cure.   Three (Nos. 5, 6, and 7), the oldest and most severely affected, were killed.

A rough *post-mortem* examination was made in one case by the owner of the dogs.   He states that the vagina was full of growth, which extended up into the uterus, forming a mass noticeable on opening the abdomen.   In another case, in which the abdomen was opened by a veterinary surgeon in the presence of the owner, the latter observed that the peritoneum was studded with small growths.

On 13th January 1896, we examined four of the bitches (Nos. 8, 9, 10, and 11) which were still affected with vaginal growths.   One of these (No. 8) had been under treatment for eighteen months, and showed slight improvement as the result of local and general treatment.

A dog, B, which had served the affected bitches (2, 10, and 11), was found on 22nd December 1895, to have what the owner described as a collar chancre about ¼-inch wide, entirely surrounding the penis just behind the corona.   This growth increased in size, and when seen by us on 19th March 1896, presented the same character as the growths affecting the bitches.

This dog had served two healthy bitches before the growth on the penis was noticed.   One of the bitches (No. 12) came in pup, and developed a growth similar to those observed in the others of the series.   The other bitch did not come in pup, and was not affected.

Shortly, then, we have the following history of infection.   Of twelve bitches, served by a dog suffering from a growth on the penis, eleven became affected with similar growths in the vagina.   Three of these bitches were served by a second dog, who subsequently developed a growth on the penis.   After becoming infected, this second dog served two healthy bitches.   One of these bitches developed a growth in the vagina, but the other remained unaffected.

All the dogs were the property of one owner.   There was consequently no difficulty in obtaining an accurate history, and no doubt whatever about the source of infection in every case.   On account of the value of the dogs, the owner has naturally taken great interest in the matter, and he has given us every facility for carefully examining the animals on many occasions.   We are not at liberty to publish the name of the owner, nor to mention the breed of the dogs.

In addition to the above series we have seen five bitches of another breed affected with similar growths.   The tumours developed after the bitches were served by a certain dog, which we have unfortunately had no opportunity of examining.   One of these bitches was subsequently served by another dog ; and, in spite of every precaution being taken in the way of frequent washing of the penis after coitus with antiseptic lotions, a number of small growths made their appearance upon the penis.   The growths in this series were exactly similar to those in the first series.

Description of Tumours.—The growths are either single or multiple, and frequently many small tumours are found at an early stage. Even when single at the commencement, infection of other parts of the mucous membrane often takes place by the time the tumours have reached the size of a mulberry.

A common situation for the tumours in the bitch is the neighbourhood of the urethral orifice ; and on several occasions we have

removed single growths from this situation. In other cases they occupy some portion of the vestibule,[1] or lie just within the vulval outlet. In one case we excised a growth the size of a mulberry from the posterior margin of the vulval orifice. When the growths are large and multiple they may completely fill the vagina, and they may extend upwards beyond the reach of the finger. These large masses often protrude from the vulva and distend the perineum.

The dog B had a pinkish, rather firm lobulated mass, from half to three quarters of an inch broad, entirely surrounding the penis behind the corona. There were, in addition, a few small growths on the glans.

In the majority of the cases we examined the growths were already of some months' duration. They then usually appear as lobulated masses, slightly constricted at the base, and of a pink or purple colour. The lobulation is sometimes coarse, sometimes fine, but the indentations are never deep. The tumours vary in consistence, being sometimes soft, sometimes firm, but never hard. As a rule, they readily bleed on manipulation by the time they have reached the size of a mulberry. On section the larger growths present a uniform whitish surface, moderately firm in consistence.

In one case we found a firm growth, about one and a half inches in diameter, almost completely smooth on the surface, hanging from the vaginal wall by a flattened pedicle, less than half an inch in its broadest diameter. This growth was microscopically identical with the lobulated sessile growths.

In another case (*see* Fig. 1) we were able to watch the progress of the disease on the penis of a dog from its commencement.

FIG. 1.

Within a week of infection the growths appeared as small glistening elevations, about the size of millet seeds, mostly transparent, but sometimes blood-stained. They looked like vesicles, but on pricking they

[1] The vestibule in the bitch is large, the urethral orifice being situated some distance from the vulval outlet.

proved to be solid.   One was removed, and on microscopical examina-
tion showed the same structure as the more advanced growths.   At
the end of two months they were still small, but were more solid in
appearance.   At the end of six months two of the growths had con-
siderably increased in size, and were about half an inch in diameter,
while the remainder had disappeared.   In a year the growths had

FIG. 2.

Case of bitch first seen in August 1896. The vagina has been cut open, and three nodules of
    growth are seen in the vestibule. The rod is placed in the orifice of the urethra. The
    vagina above the urethral orifice is free from growth.   (From a drawing by Dr T. G.
    Stevens.)

further increased in size, and had assumed the usual lobulated appear-
ance.

In the majority of cases there was no deep induration, and the
growths were readily removed from the subjacent structures with the
mucous membrane to which they were attached.   In two cases in

which we had an opportunity of making a *post-mortem* examination there was infiltration of the deeper tissues.

The first case was that of a bitch which was first seen by us in January 1896, when the vagina was filled by a large mass of growth which distended the perinæum. In August the growth had diminished in size, and presented at the vaginal orifice as a firm, warty, irregular mass, exactly resembling a malignant tumour. The perinæum was partially destroyed, and the vulval outlet was represented by an irregular opening, one and a half inches long and one and a quarter inches broad, with hard everted edges. In December the opening was two inches long and one and a half inches broad, and the growth had extended in the subcutaneous tissue for about an inch from the margin of the orifice as a firm nodular mass, more or less adherent to the overlying skin. The animal succumbed some days after an operation for the removal of the growth. At the *post-mortem*
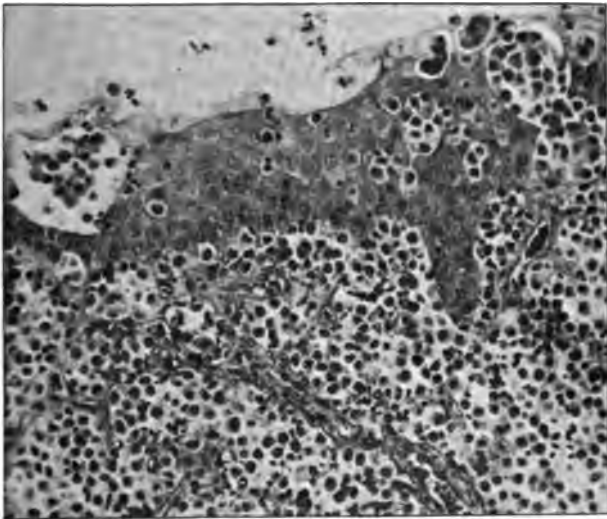
FIG. 3.

Section through one of the tumours. On the surface the epithelial lining of the mucous membrane is seen; the mass of the growth is composed of cells with round nuclei, enclosed in irregular alveolar spaces.

examination the kidneys were found to be affected with interstitial nephritis, and no secondary growths were found. The tumour was carefully examined, and it was found to have infiltrated the vaginal wall. It had the same microscopical structure as the other growths.

The second case was that of a bitch which was first seen by us in August 1896, when there was a growth about an inch in diameter close to the urethral orifice, apparently similar to the growths in the other dogs. In October of the same year she succumbed to puerperal septicæmia, shortly after pupping. At the *post-mortem* examination we found, on the left of the urethral orifice, an irregular mass, one and a half inches in diameter, torn and ragged in the centre as if injured during parturition. On cutting through the base of the tumour

it was seen to extend deeply as a firm, whitish, lobulated, well-defined growth, which infiltrated the muscular wall, and which could not be shelled out. Two rounded tumours, half an inch in diameter, were found beneath the mucous membrane of the vestibule, near the vaginal orifice. Over each of these there was a slit in the mucous membrane, through which a small mass of growth fungated. These tumours had been noticed about two months before death, and they had been shelled out through incisions made in the mucous membrane; but they had recurred, and had fungated through the incision. On section the masses were found to infiltrate the vaginal wall. They were round, yellowish-white in colour, with a well-defined outline. Although encysted, the growths could not be completely shelled out. The glands in the groin were enlarged. No secondary deposits were found in the viscera.

The dog which served this bitch became infected with a number of small growths on the penis. These we removed, and on microscopical examination they were found to be similar to the rest of the growths. Up to the present no recurrence has occurred.

Microscopical Appearances of the Growths.—Covering the free surface of the tumours is a layer of stratified epithelium, continuous with and similar to that lining the vagina. The epithelial layer is frequently thinned out, but is never absent. Corresponding to the slight indentations between the lobules, the epithelium dips in for a short distance. The epithelium is separated from the subjacent tissue by a layer of connective tissue, sometimes very delicate, but always demonstrable by appropriate staining methods.

The main mass of the growth is made up of cells, which are very regular in size, and possess round nuclei. In some of the tumours— apparently the more rapidly growing ones—the cells are loosely packed, preserving their rounded forms. In others they are closely packed, and assume a polyhedral shape, giving at first sight the impression of an epithelial new formation. This is especially the case in those growths where the stroma is relatively large in amount, and encloses the cells in irregular alveolar spaces.

The stroma varies considerably in amount in different growths. In some, especially in those which are increasing rapidly, it is very scanty; in others it is relatively well marked. The alveolar arrangement mentioned above is especially marked just beneath the surface epithelium, the general structure and appearance being very similar to the alveolar sarcomata of the skin in the human subject.

Numerous small thin-walled blood-vessels may be seen, not only in the supporting framework, but also between the cells.

In the two cases in which the growths infiltrated the vaginal wall and the perinæum the structure was identical with that just described.

So far as the structure is concerned the growths are to be regarded as sarcomata. They differ entirely from the venereal tumours met with in the human subject, which result mainly from an epithelial overgrowth.

Dr Plimmer kindly examined some of the tumours, and was unable to demonstrate with certainty the presence of the protozoa described by himself and Professor Ruffer in malignant tumours.

We have hitherto failed to find any micro-organisms which we can claim to bear a casual relation to the tumour. We hope, how-

ever, to make further observations in these directions at a later date.

Secondary Deposits.—We have found no secondary deposits in the viscera ; but our *post-mortem* examinations have been limited to two cases, in neither of which has the animal died from the direct effect of the disease. The owner of the dogs, however, informs us that in one case he noticed tumours on the peritoneum after death.

In one case we found the inguinal glands enlarged. Microscopical examination showed that they were infiltrated with groups of cells, similar in size and shape to those of the primary growth.

Course taken by the Tumours.—We have in no case observed a spontaneous cure. The growths have always gradually increased in size, and fresh tumours have appeared on the vaginal wall, or on the penis. The owner thinks that this occurs especially after hæmorrhage caused by manipulation, and after unsuccessful attempts at removal. The rate of growth is slow at first, but becomes more rapid at a later period, so that by the end of a year or eighteen months the vagina is completely filled by a mass as large as an orange.

In the early stages the growths are confined to the mucous membrane, and do not infiltrate the deeper parts. In the course of time, however—perhaps some two or three years from the commencement of the attack,—infiltration of the deeper parts takes place. In the highly bred class of dogs under our notice there seems to be a tendency for the growths—originally simple in character—to take on a malignant course, leading directly or indirectly to death.

The Relationship of the Growths to Discharge.—The dependence of venereal warts in man upon the continued irritation or venereal discharge has for a long time been a prevailing belief, and we find that a similar view is held by some veterinary surgeons with regard to the causation of papillomatous growths on the genitals of dogs (*e. g. vide* Kitt).

Recently C. W. Cathcart[1] has brought forward evidence to show that venereal warts in man may be conveyed independently of a gonorrhœal or syphilitic discharge. He believes that venereal warts are specific and contagious, and that they are associated with, but not dependent upon venereal discharge.

We have gone carefully into this question in the case of the dogs, and we feel quite certain that the tumours are not dependent upon irritation due to discharge.

When the dogs were first brought under our observation we noted especially the fact that, with the exception of the cases which were being treated with caustic, there was an absence of purulent discharge. The growths, however, which we first examined were of large size, and we thought that there might have been an antecedent discharge, which had ceased before the dogs came under our observation. We consequently examined the dogs in which the growth was in an early stage. In no case did we find any purulent discharge.

In the case which we successfully inoculated the growths were not preceded by discharge.

[1] Journ. Path. and Bacteriol., Edin. and London, July 1896.

The owner from his own observations states that the growths are unattended with any purulent discharge. The first thing noticed in the bitches was a slight sanguineous discharge some time after being served ; and, unless the growths were specially looked for, this bleeding was the first evidence of their existence.

Treatment.—After watching the results of various modes of treatment, we have no hesitation in saying that, when possible, the tumours should be excised. In most cases this is readily accomplished by snipping the mucous membrane around the base of the tumour, and stripping it off with the attached growth from the underlying tissues. Some vessels may require ligation, and the edges of the mucous membrane may then be brought together with a few stitches. We have operated in this way upon six occasions, and there has been no recurrence of the growths. In one case it was necessary to divide the perinæum, in order to get at a large mass of growth 3 inches in diameter, situated on the anterior wall and the sides of the vagina. The perinæum was then sewn up, and the mucous membrane, as far as possible, loosely brought together with stitches. The wound healed well, and there has been no appreciable contraction of the outlet.

When the growth has infiltrated the vaginal wall the only possible method of treatment is by excision of the part of the vagina affected. In one case we excised about 4 inches of the vagina, but without success, death apparently resulting from a chronic affection of the kidneys. Partial removal by tearing away the tumour with forceps is useless, and only leads to a rapid extension of the growth.

The destruction of the growth by the use of caustics is a long and troublesome process, and unsatisfactory in the extreme. Much cicatricial tissue forms, contracting the lumen of the tube ; and even after the prolonged use of caustics it is difficult to be certain of a complete cure.

Inoculation Experiments.—We have not yet completed our inoculation experiments, so that we will only mention briefly the main results. We hope at a later date to publish a full and detailed account.

Attempts to infect rabbits and guinea-pigs have completely failed.

A dog which was inoculated on the penis developed a typical growth which, after some months, had reached the size of a marble.

A minute portion of a tumour removed from the vagina of a bitch was placed in the subcutaneous tissue of the abdomen of a dog. In a fortnight a distinct, nodulated, well-defined tumour, half an inch in diameter, had appeared in the subcutaneous tissue at the seat of inoculation. The tumour gradually increased in size, and at the end of two months the animal was killed by chloroform. At the *post-mortem* examination a well defined nodulated tumour, $1 \times \frac{1}{2}$ inch, was found in the subcutaneous tissue. On section it had a pinkish-white appearance, and a microscopical examination revealed the same structure as the rest of the growths.

In another case, after inoculation into the peritoneal cavity, a nodule appeared in the abdominal scar. At the end of two months this nodule formed a projecting lobulated mass, $1\frac{1}{4}$ inches long, and

¾ inch broad. A month later the tumour had almost completely disappeared, and the animal was killed.

At the *post-mortem* examination a nodule the size of a pea was found imbedded in scar tissue.[1]

Remarks.—There seems to be no doubt that the tumours in question are of the same nature as those described by veterinary surgeons under the names of condylomata, papillomata, or warts; but we cannot find in veterinary works any account of microscopical appearances, nor any suggestion of infectivity.

They also appear to be identical with the tumours described by Wehr, Geissler, and by Duplay and Cazin, in their experiments upon the infectivity of cancer; so that we think it well to give a short abstract of their work.

Geissler[2] was successful in inoculating from a cauliflower-like tumour occurring on the prepuce of a dog. The tumour consisted of an alveolar framework enclosing masses of polymorphous cells which were closely packed together in some alveoli, and loosely packed in others. Although he considered the tumour to be carcinomatous, he stated that it differed in structure from the typical carcinomata found in the human subject. In the discussion which followed his paper the general expression of opinion was that the growth was not a carcinoma, but that it consisted of a mixture of granulomatous and sarcomatous tissue.

He inoculated a series of dogs with pieces about the size of a millet seed, taken from the deeper part of the tumour, and in two cases the inoculation proved successful.

A bitch was inoculated in the subcutaneous tissue of the abdomen, the material being pushed into the deeper part of the tissue by means of a trocar and canula. In three weeks a tumour the size of a plum formed, but it ultimately disappeared. Portions of the tumour removed during life showed the same structure as the original growth.

A dog was inoculated in the same way in the subcutaneous tissue of both flanks, and also in the cavity of the tunica vaginalis. In five weeks a tumour appeared in each of the three places. One of the tumours in the flank ulcerated. The animal died at the end of eight months. At the *post-mortem* examination six tumours, each about the size of a bean, were found in the wall of the thorax ; some were adherent to skin, and others freely movable. In the linea alba two ulcers with indurated bases were found, the one $6\frac{1}{2} \times 3\frac{1}{2}$ cm., and the other $2\frac{1}{2} \times 1\frac{1}{2}$ cm. In the scrotum there was a tumour about the size of a pea adherent to the skin, but freely movable over the underlying tissue. On the left side of the prepuce there was a hard swelling adherent to the skin, but movable over the penis. The lymphatic glands all over the body were enlarged, especially those in the neck. Many small secondary deposits were found in the skin over the inner side of the thigh. On opening the abdomen the left spermatic cord was thickened, and contained a tumour about the size of a cherry, compressing the ureter. There were many small nodules in the spleen, liver, and peritoneum. On the parietal pleura there was a villus-like growth. Unfortunately, on account of an accident with the preserving fluid, no microscopical examination was made of

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the secondary deposits, and consequently their real nature remains
doubtful. A portion of one of the tumours at the seat of inoculation
was removed during life, and showed the same structure as the
original growth. With this portion of tumour a third dog was suc-
cessfully inoculated ; and at the time of publication of his paper the
resulting tumour was still increasing in size.

Wehr[1] made a series of inoculations with what he considers
undoubted carcinomata. The tumours in question affected the pre-
puce and the vestibule of the vagina of dogs in the form of
papillomatous, cockscomb-like, soft, medullary growths. He states that
they are similar to the growths described by veterinary surgeons as
condylomata. Histologically they consisted of a scanty stroma,
containing many cells with round nuclei closely pressed together.

Altogether he inoculated twenty-six dogs in the subcutaneous
tissue, using the same method as Geissler. In most of the cases
tumours appeared at the spot of inoculation, but disappeared in six
or eight weeks. Portions of some of the tumours removed during life
showed the same structure as the original growth.

In the case of one bitch, which was inoculated in four places, death
ensued at the end of seven months. At the *post-mortem* examination,
in addition to a tumour the size of a plum at the site of inoculation,
there was a tumour the size of an apple in the retro-peritoneal glands
on each side of the vertebral column, compressing the urethra, and
causing rupture of the bladder. There were two enlarged glands in
the thorax, and a few scattered nodules in the spleen. Wehr states
that these secondary deposits were of a carcinomatous structure.

Duplay and Cazin[2] made an extensive series of inoculations in
order to study the infective nature of tumours. In all cases of
undoubted malignant growths the results were negative. They, how-
ever, performed successful transplantations to the genitals of dogs
from a growth the size of a walnut occurring in the vagina of a bitch.
They state that the microscopical character of the tumour resembled
inflammatory rather than carcinomatous tissue. The tumours which
developed after inoculation were about the size of hazel-nuts, and
were of similar structure to the original growth. In one of the
successful cases there was found, in addition to a tumour on the penis,
at the site of inoculation a deposit of an epithelial character in the
testis. They, however, consider it possible that this deposit bore no
causal relation to the inoculation.

The tumours described by all these observers resemble those in our
series, both in their situation on the genitals of dogs and in their
naked-eye appearances. From the descriptions given we believe, too,
that they are similar in microscopical structure. We differ, however,
in our views about the nature of the tumours. Duplay and Cazin say
that the structure resembles inflammatory rather than carcinomatous
tissue. Geissler and Wehr consider the tumours to be carcinomata.
We have already stated that whilst the alveolar arrangement of poly-
hedral cells in some of the tumours may suggest at first sight an
epithelial new formation, there is nothing in the microscopical
structure of the tumours, either at an early or at a late stage, to
distinguish them from ordinary round-celled sarcomata.

[1] "Deutcher Chir. Cong.," 1888 and 1889.
[2] Trans. Eleventh Internat. Med. Cong. in Rome, vol. ii., p. 103.

Both Geissler and Wehr noticed that in some cases tumours which had developed after subcutaneous inoculation, ultimately disappeared, although portions of these tumours, when removed, showed the same structure as the original growth. This agrees with our own experience above quoted.

Geissler and Wehr appear to have been successful in obtaining secondary deposits in the viscera. We have, up to the present, no conclusive evidence of the occurrence of secondary deposits in the organs ; but the infiltration of the deeper tissues in two of the cases, and the affection of the glands in one case, show that the tumours are capable of assuming a malignant course.

Conclusions.— 1. The tumours we have described affect the genitals of dogs, and are probably identical with the papillomata, condylomata, and warts of veterinary surgeons, and with the infective tumours described by Geissler, Wehr, and by Duplay and Cazin.

2. The contagion is conveyed during the act of coitus, and the tumours are not dependent upon the irritation of any discharge.

3. The tumours can be transplanted artificially, not only to the mucous membrane of the genital organs, but also to the subcutaneous tissue.

4. The muscular wall of the vagina may be infiltrated, and secondary deposits may occur in the lymphatic glands.

5. The clinical identity of the infiltrating tumours and the simple outgrowths is shown by the case in which a bitch with an infiltrating tumour infected a dog with multiple simple outgrowths.

6. The structure of the tumours is identical with that of a round-celled sarcoma.

7. The tumours which have developed in the subcutaneous tissue after inoculation may disappear in the course of a few months.

ANTHRAX.

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Definition.—Since the discovery of the anthrax bacillus and the demonstration of its pathogenic properties, it has been possible to give an exact definition of the disease which is now generally known in this country under the name of anthrax. That word must now be reserved for the malady which is caused by the anthrax bacillus. Every case of disease determined by that organism—no matter what may be the symptoms manifested or the lesions induced—must be called anthrax, and no case of disease that has any other cause must be so named, no matter how closely it may resemble a case of anthrax in respect of symptoms and lesions.

Furthermore, it is very undesirable that the word anthrax, even when qualified by an adjective, should be applied to morbid conditions that are etiologically distinct from the disease caused by the anthrax bacillus. On this account the use of the words anthracoid and symtomatic anthrax ought to be avoided. The first of these is as meaningless as "glanderoid" or "tuberculoid," and the latter tends to promote confusion by suggesting a relationship between two diseases that are etiologically quite distinct.

Discovery of the Bacillus.—The first published observation regarding the occurrence of rod-shaped bodies in the blood of animals dead of anthrax emanated from Rayer and Davaine in 1850, but Pollender is sometimes credited with the discovery because, in an article which appeared in 1855, he claimed to have observed the rods in 1849. Neither Davaine nor Pollender in recording the discovery claimed that these rods were etiologically connected with the disease in the course of which they were found, but in 1863 the previously published researches of Pasteur on butyric fermentation led Davaine to definitely declare the opinion that the rod-like bodies which he had first seen in 1855 were the actual cause of anthrax. The final and decisive proof of the correctness of this view was not furnished till 1876, when Koch discovered the phenomenon of sporulation on the part of the anthrax rods, and demonstrated the infectivity of artificial cultures.

Morphology of the Bacillus Anthracis.—In the blood of an animal recently dead anthrax bacilli occur in the form of cylindrical, straight or slightly curved, motionless rods, about $1\cdot5\,\mu$ in breadth, and varying in length from $4\,\mu$ to $20\,\mu$. When unstained these rods appear structureless, and they are generally termed "bacilli," as if they represented the elementary units or individuals of the species. That, however, is a mistake. All but the very shortest rods found in the blood when appropriately stained are found to be composed of from two to ten or more segments, which are the true "bacterial cells" or individual elements. The segments of a rod are held together by a common envelope, and united end to end by a thin disc which appears to be identical in composition with the envelope.

The appearance of the rods varies according to the method of staining. When a cover-glass preparation of anthrax blood is stained with an aqueous solution of methylene blue, washed in water, dried, and mounted in Canada balsam, the rods generally appear considerably thinner than when they are stained in the same way with an aqueous solution of gentian violet. That is because the latter stain attaches itself to the envelope as well as to the central protoplasmic part of the bacterial cells, while the methylene blue leaves the envelope uncoloured and invisible. With either of these methods of staining the ends of the bacilli and the ends of their segments appear square cut, or even slightly concave. In the latter case the unstained material between the ends of the segments takes the form of a minute biconvex lens. With deep gentian-violet staining, however, the thinner septa as well as the envelope are stained, and many of the segments thus appear longer than they really are.

The best method of demonstrating the envelope of anthrax bacilli is that recommended by Johne. It consists in staining with 2 per cent. aqueous solution of gentian violet, washing in water, then dipping for a few seconds in water acidulated with acetic acid ($\frac{1}{2}$ to 1 per cent.), and washing again in water. The cover-glass thus stained is dried on its upper face, and then mounted for examination, not with Canada balsam, but with a drop of water. By this method of staining the axial protoplasmic substance of each segment is stained a deep violet, while the envelope and the septa between the segments retain merely the faintest tinge of violet colour, or even without that are visible, owing to their having a refractive index different from that of the water in which they are examined (Plate I., Fig. 3). Every septum

being here unstained, the segments may appear shorter than when the accidulated solution is omitted, and the ends of the rods and segments no longer appear concave, but are either flat or distinctly convex.

The presence of a thick easily demonstrable envelope to the bacilli is, as Johne has insisted, of great value in distinguishing between the rods of anthrax and putrefactive bacteria with which they may be associated in blood that is not quite fresh. A further valuable distinction in such cases is afforded by the fact that with simple aqueous solutions of the basic aniline dyes, and particularly with methylene blue, anthrax bacilli stain less intensely than the bacilli of malignant œdema and most other putrefactive bacteria that approach the former in size and shape. This difference of tint is quite distinct even when the comparison is made between anthrax bacilli in fresh blood and the malignant œdema bacilli of putrid blood, and it is much more evident when the comparison is made between the different bacilli in the same preparation, for in blood invaded by putrefactive bacteria anthrax bacilli have, in consequence of degeneration, in considerable measure lost their staining affinities.

The bacillary or rod-like form is the common one in the blood and spleen pulp, probably because the blood current tends to break segments off the longer rods, and thus interferes with their natural tendency to grow out into the filamentous or leptothrix form. Occasionally, however, filaments of considerable length are found in the body, rarely in the blood, but usually in extra-vascular positions, such as the gelatinous œdema of a local lesion, or in the tissue of a lymphatic gland.

A point of great importance in connection with anthrax bacilli as they occur in the blood or tissues of the living animal, or in these situations in the intact carcase, is that they never proceed to the development of spores.

When blood bacilli are transferred to suitable artificial media, kept at a proper temperature and exposed to the air, they give rise to cultures in which the filamentous form of the organism predominates. These filaments are produced simply by the undisturbed growth of the shorter rods, and they may attain a great length. When examined unstained such young filaments appear glassy and structureless, like the unstained rods found in the blood, but when appropriately stained they are found to have a structure identical with that of these rods, save that the enveloping sheath is often thin or scarcely demonstrable. The existence of the envelope in the filaments of artificial cultures has even been denied, but erroneously, as Fig. 5 in Plate I. will show. As in the case of the blood bacilli, the length of the segments or bacterial cells varies in different cultures.

The segments of the rods and filaments represent the vegetative cells of the anthrax organism, and, strictly speaking, it is only by their growth and division that the germ multiplies. A single segment grows until it has attained a size approximately double that with which it started, and then by the formation of a septum across its centre it is divided into two new segments. These may remain connected, until, by repetition of the process of fission, a rod or a filament is built up, or they may separate and give rise to independent rods.

This division by fission is the only method of reproduction that takes place in the blood and tissues of the living animal, and in certain circumstances it is the only method of growth in artificial cultures

also. But when either anthrax blood or artificial cultures are freely exposed to the air and kept at a suitable temperature the phenomenon of sporulation sets in, and culminates in the formation of spores, which bear to the vegetative cells of the bacilli a somewhat similar relationship to that which seeds bear to the higher plants.

Sporulation.—The process of sporulation may without trouble be followed under the microscope in a hanging-drop preparation kept at a temperature approaching that of the body. The segments of a filament that are preparing to sporulate lose their hyaline aspect, their substance becoming granular and more opaque. Towards the centre of the segment a bright refractile speck now appears, and continues to grow at the expense of the original axial or protoplasmic substance of the bacterial cell. This is the spore, and when it has attained its full size it is oval in shape, with its long axis coinciding with that of the segment. When the spore has reached its full size it is set free by the dissolution of the last remnants of the bacterial cell from which it was formed.

The mode of formation of spores may also be studied by examining stained cover-glass preparations of sporulating cultures. With simple aqueous solutions of the basic aniline dyes, used at ordinary temperatures, the spores remain unstained, and in sporulating filaments appear as oval clear specks in the stained substance of the segments (Plate I., Fig. 5). Not more than one spore is formed in a single segment.

The possession of this power of sporulation is of immense advantage to the anthrax germ, as it enables it to maintain its existence in the stress of circumstances that would otherwise be promptly fatal to it. This arises from the fact that the spores offer much greater resistance than the vegetative elements or bacilli to such agents as heat, sunlight, and chemical disinfectants.

The circumstances necessary for sporulation are (1) *free* exposure of the anthrax rods or filaments to the air or oxygen, and (2) a temperature between 18° and 42° C. (64° and 107° F.). The necessity for a free exposure to oxygen explains why sporulation never takes place in the blood or tissues during life, or in these after death as long as the carcase remains intact. With a suitable temperature, one has only to shed the blood, or incise the skin, and the bacilli that are thus exposed to the air, or some of them, will at once begin to sporulate. The influence of oxygen on sporulation is also seen in artificial cultures. Provided the temperature is suitable, these always sporulate abundantly when the growth is a surface one, as on potato or agar, or when it is effected in a thin stratum of liquid. On the contrary, sporulation is scanty or entirely absent in the filaments grown at the bottom of a deep stratum of liquid, even although the temperature be quite suitable.

The temperature at which sporulation is most rapidly effected is from 90° to 100° F. In artificial cultures grown at these temperatures, with free exposure to the air, many filaments have resolved themselves into spores in twenty-four hours. At 65° to 70° F. sporulation requires about a week, and the rapidity of the process between 70° and 90° is proportional to the temperature. As will presently be shown, these facts are of great practical importance.

In the dried state and protected from sunlight, anthrax spores retain their vitality and power of germination for an indefinite period,

but as soon as they find themselves in suitable conditions of moisture and temperature they germinate and produce vegetative elements or bacilli.

In the process of germination the protoplasmic centre of the spore gives rise at one of its poles to a minute bud, which soon assumes the form and dimensions of one of the before-described bacterial cells, and this, in suitable conditions, multiplies by fission and leads to the development of a fresh cycle of bacilli. The protrusion of the protoplasmic bud is apparently made possible by the gradual absorption of the external envelope of the spore at the spot where the bud is to issue.

Staining of the Bacilli.—The anthrax bacillus is an easily stained organism. In cover-glass preparations of blood, tissue juices, or artificial cultures, it instantly takes the colour of any of the basic aniline dyes in aqueous solution. It is also stainable by the method of Gram, but requires a little more care to avoid decoloration than in the case of some other organisms, such as the pyogenic cocci.

As already mentioned, the spores of anthrax remained unstained when treated with simple aqueous solutions of the basic aniline dyes at ordinary temperatures. This difficulty of staining the spores appears to be due to their resistant external envelope, and it may be overcome by using carbol-fuchsin solution at a temperature of 120° C. (in the autoclave). The substance of the filaments in which the scarcely mature spores are set may afterwards be contrast-stained with aqueous solution of methylene blue. In successful preparations obtained in this way the bright red oval spores occur at intervals along the course of the blue-stained filaments.

Cultivation.—Within the limits of temperature required for its growth, the anthrax bacillus may be cultivated in a great variety of animal and vegetable infusions, provided these are neutral or slightly alkaline in reaction and are exposed to the air. The temperature limits are 16° and 44° C. (60° and 111° F.) At the lower limit growth is very slow, and at temperatures above 43° C., it is also meagre and soon ceases, the bacilli perishing without having formed spores. Growth is most rapid and luxuriant between 32° and 38° C. (90°-100° F.).

The anthrax bacillus is a strict ærobe. Like animal cells it requires for its growth oxygen that is either free or only loosely combined (as in the case of the red blood corpuscles). In the complete absence of oxygen no growth takes place, and the withdrawal of oxygen from growing cultures within a comparatively short period determines the degeneration and death of the bacterial cells, though it leaves the vitality of the spores unimpaired. In consequence of its ærobic habit the growth and multiplication of anthrax bacilli in the blood and tissues of the body ceases as soon as death takes place.

The following are the principal characters of the anthrax cultures in the nutritive media in common use in the laboratory.

In broth contained in test-tubes the growth takes the form of a fine "fluffy" material, which, if the liquid is not disturbed, entirely settles to the bottom of the tube, and leaves the supernatant liquid quite transparent.

In gelatine plate cultures the colonies develop as white opaque round grains, which when magnified are found to be composed of

intricately interwoven leptothrix filaments. The course of these filaments can only be distinctly made out at the "frayed out" surface of the colonies (Plate I., Fig. 6).

In gelatine stab cultures a white line of growth develops along the needle track, and from it processes like thistledown shoot out laterally into the gelatine. Liquefaction of the gelatine then begins at the surface, and slowly proceeds until the whole mass down to the bottom of the needle track has been liquefied.

On the surface of agar the growth is white and shining, resembling "frosted" silver or hoar frost when held between the eye and the light.

On potato the growth is luxuriant, dull white in colour, and opaque.

In all these artificial cultures the organism grows in the form of leptothrix filaments, which in the before specified conditions proceed to sporulation.

Resistance of the Virus.—In considering the resistance offered by the anthrax germ to destructive agents, it is necessary to distinguish between the spore and the spore-free bacillus.

Fresh anthrax blood, which when it is enclosed within the body or has only recently been shed contains only bacilli, is rendered innocuous by prolonged desiccation (over sixty days at ordinary temperatures), but desiccation is without effect on anthrax spores. Sunlight is fatal to the bacilli in blood exposed to the air in less than twenty-four hours, but, in the dry state, spores may resist exposure to sunlight for a month or more.

The bacilli in fresh anthrax blood are killed by a few minutes' exposure to a temperature of 60° C. A moist heat of 100° C. is fatal within five minutes to spores, but they may resist a temperature of 95° C. for ten minutes. To instantly destroy spores in the dry state, a temperature of about 160° C. is required.

Bacilli in blood or culture are quickly destroyed by exposure to 2½ per cent. solutions of carbolic acid or chloride of lime in water, or by ·2 per cent. aqueous solution of mercuric chloride. On the other hand, spores are not with certainty destroyed by a week's exposure to a 5 per cent. solution of carbolic acid in water, but ½ per cent. solution of mercuric chloride is fatal to them in a few minutes when contact can be assured, as in the case of artificial cultures.

Attenuation of Virulence.—Speaking generally, it may be said that the virulence of anthrax bacilli, as they are found in the blood of animals or in artificial cultures, may be diminished by exposing them for somewhat less than the fatal period to an agent that is destructive to them. Thus when fresh defibrinated anthrax blood is heated to a temperature of 55° C. for ten minutes, some at least of the bacilli are not destroyed, but are so modified that the defibrinated blood in quantities of 3 cc. may be used as a protective vaccine for sheep (Toussaint). In this case, however, the modification impressed upon the organism is transient, and not transmitted to succeeding generations if these are grown under ordinary conditions. On the other hand, more or less permanent diminution of virulence, transmissible to succeeding generations, may be impressed upon bacilli by cultivating them under conditions that are somewhat unfavourable to growth and prevent sporulation.

An attenuated virus has thus been produced by the action of compressed air or oxygen on growing cultures (Chauveau), by cultivating

the organism in artificial media containing minute quantities of carbolic acid or potassium bichromate (Chamberland and Roux), or by maintaining artificial cultures at a temperature approaching the highest point at which growth takes place. The last of these methods was devised by Pasteur, and employed by him to prepare cultures so attenuated that they might be used as vaccins to protect animals against natural attacks of the disease.

In the preparation of these vaccins the anthrax bacillus is cultivated in bouillon at a temperature between 42° and 43° C. By maintaining the temperature slightly over 42° sporulation is prevented and that is absolutely essential in order to effect attenuation., Incubation for about a month in these conditions entails the death of the culture, but short of the period necessary to destroy the vitality of the organism such incubation renders it less and less virulent. After from fifteen to twenty days' incubation the culture is still fatal to mice, but harmless to guinea-pigs and rabbits, and it is then suitable to use as a " first vaccin." The " second vaccin " is less attenuated, since it will kill guinea-pigs as well as mice, and make rabbits ill. This degree of attenuation is obtained by incubating in the conditions mentioned for ten to twelve days. When the bacilli of these attenuated cultures are used to start fresh cultures incubated at 37° or 38° they yield spores, and these again may be employed to start further cultures which retain the attenuation although grown at ordinary temperatures.

Distribution of the Bacilli within the Body.—As a rule, which has few if any exceptions in the case of the domesticated mammals, when an animal succumbs to anthrax the blood throughout the whole body contains the bacilli. These are most numerously present in the capillaries, but they are also found in great numbers in the blood of the veins, both large and small. The spleen pulp also contains immense numbers of them. In some situations, such as the renal glomeruli, the lungs, and the intestinal villi, the capillaries are generally crammed with the bacilli to such a degree that in stained preparations these vessels appear as if they had been filled with a coloured injection.

Anthrax is thus generally a veritable septicæmia, that is to say, a disease in which the blood is the main seat of propagation of the causal organism. For the most part the tissues outside the vessels are free from bacilli, but there are always exceptions to that rule. In the first place, the rods may be found in extra-vascular positions as the result of minute hæmorrhages. They thus find their way into the uriniferous tubules and urine, a fact which is of great importance from its bearing on soil contamination. When the primary seat of the disease is the intestine, bacilli are also found in the lumen of the bowel, from which they may be passed out with the fæces. In the second place, bacilli are always found outside the blood-vessels in the neighbourhood of the point at which they first found a foothold in the body. They are thus always found in the inflammatory exudate of the local swelling at the seat of inoculation, in the tissues of the throat and its lymphatic glands when the primary point of infection is the mucous membrane of the fauces or pharynx, and in the intestinal wall and the mesenteric or other lymphatic glands of the bowel when the intestinal mucous membrane is the part first invaded.

·Even in the most susceptible species—those in which the disease most rapidly becomes septicæmic—anthrax is thus to begin with a local disease, in which the bacilli are propagating in the lymphatic spaces and vessels of the part or organ first invaded.   In the case of susceptible animals the bacilli, however, soon gain the nearest lymphatic glands, pass these, and reach the blood.   On the other hand, in animals that offer a notable degree of resistance to the disease the invading organisms may for a considerable period be confined to the neighbourhood of their point of entrance into the tissues, or entirely destroyed there.   In the one case the fatal ending is materially delayed; in the other the animal recovers.

It follows from what has been said that if an animal already showing symptoms of anthrax, such as swollen throat and elevated temperature, be killed, few or no bacilli may be found in its blood.   It is also conceivable that an animal in which the throat is the part first invaded may die from asphyxia before the general invasion of the blood stream has had time to take place, but experience justifies the statement that this is a very rare occurrence in the lower animals, and the writer has never seen an indubitable instance of it.

The foregoing considerations also show why it is, that, except during a short period immediately preceding the fatal ending, the bacilli cannot by microscopic examination be found in blood taken from the living animal.

Habit of Life of the Bacillus.—There can be no doubt that in perfectly natural circumstances the anthrax bacillus frequently finds all the conditions essential for its multiplication external to the animal body, and it must therefore be classed with the facultative parasites. At the same time it is probable that at least in temperate climates, such as ours, it depends for its continued existence mainly on its power of propagating in the body.   In other words, it is probable that if the germs of anthrax which are at present in existence were permanently cut off from every opportunity of invading the animal body the species would be unable to maintain its struggle for existence in the outer world.   As has already been seen, the conditions necessary for its growth are (1) a suitable nutritive medium, (2) a temperature between 60° and 111° F., and (3) the presence of free oxygen.   But when it is growth external to the body that is being considered a fourth condition must be recognised as essential, viz., the absence of more rapidly growing purely saprophytic bacteria, such as those of putrefaction.   When anthrax bacilli are voided in urine or excrement in the summer months they find themselves in the presence of the conditions necessary for growth and multiplication, in respect alike of nutriment, temperature, and exposure to oxygen. But if the temperature is only a few degrees above 60 they are unable to sporulate, and their rate of multiplication is slow.   They are therefore almost certain to be crowded out of existence in the struggle with other organisms that grow much more rapidly at the same temperature.   Another factor unfavourable to the bacilli is desiccation, which first arrests the growth of the bacilli, and finally destroys their vitality.   There are therefore good grounds for believing that during the greater part of the year in this country surface contamination of the soil with blood, urine, or excrement of an anthrax animal is only temporary.   If, however, the temperature is over 70° F., the result

may be very different, for the bacilli may then resolve themselves into spores, and these are capable of resisting the most adverse conditions that they are likely to encounter in a natural way in soil or water.

These considerations show that the anthrax organism leads a very precarious saprophytic existence, and make it probable that most outbreaks of the disease in this country are ascribable to the ingestion of spores or bacilli that are separated by only a few generations from the bacilli voided from the body of an animal suffering from anthrax, or that have themselves been liberated from the dead body of such an animal.

The part which is played in soil contamination by the careless burial of anthrax carcases has been much discussed. Pasteur thought that it was the main method by which the disease is spread, and he ascribed to earth-worms an important rôle in this connection, maintaining that they brought to the surface of the ground spores which had been formed in the bodies of buried anthrax carcases. But while it is impossible to deny that earth-worms thus may be instrumental in bringing anthrax spores to the surface of the ground when these do exist in anthrax graves, it is permissible to maintain that the view which Pasteur held regarding the dangers of careless burial and the rôle of earth-worms was an exaggerated one. In the first place, it ought to be remembered that in such portions of an anthrax carcase as are not in direct contact with the air the bacilli soon perish without forming spores, even although the temperature is suitable for that process. In the second place, the prompt burial of an anthrax carcase at a depth of a few feet withdraws from the bacilli two of the conditions essential for sporulation, viz., exposure to oxygen and a temperature above 64° F.

To sum up what has been said under this head, it appears highly probable that those parts of the earth's surface at which anthrax germs at present exist owe their contamination to the excrement, urine, or other discharges of animals suffering from anthrax, or to the blood shed on the surface of the soil during *post-mortem* operations.

Methods of Infection.—Anthrax may be experimentally set up in susceptible animals by inhalation, inoculation, or ingestion. It is doubtful whether the first of those is ever the cause of natural cases of anthrax in the lower animals, but the so-called "wool-sorter's disease" is an example of that method of infection in the human subject. Inoculation is also a rare method of infection in the lower animals. On the other hand, the great majority of cases of anthrax in man are contracted in this way, either during operations with fresh anthrax carcases, or with the hair, wool, or hides of animals dead of the disease. Either spores or bacilli suffice to infect in this way when implanted as deep as the dermis, and the slightest scratch or abrasion of the skin may furnish all the wound that is necessary to bring about infection if the germs of the disease are brought into contact with the part.

Undoubtedly the common method of infection in the lower animals is ingestion. The infective material may be in the form either of bacilli or spores. In the case of the herbivora it is probable that nearly all cases are caused by the ingestion of anthrax spores adhering to the food which they eat or suspended in the water which they drink. Since gastric digestion is capable of destroying the bacilli

but not the spores, infection is much more certainly induced by feeding experimentally with the latter than with the former, but the tissues of the alimentary canal anterior to the stomach are equally susceptible to infection with bacilli or spores. It is impossible to say with certainty whether some abrasion or defect of the mucous membrane is necessary for infection, and the point is of no practical importance, since it is probable that no animal at any time has the lining membrane of its alimentary canal absolutely devoid of epithelial defects. At the same time it is not open to doubt that actual wounds and macroscopic abrasions of the buccal or pharyngeal mucous membrane must carry with them extra risk of infection when the animal ingests contaminated food, and Pasteur found that a larger proportion of sheep contracted the disease when the infective material was sprinkled on such prickly materials as barley awns than when it was given on soft green food.

Probably the only natural method of infection in the pig and dog is the ingestion of raw flesh from some animal dead of anthrax. In animals of these species the mouth, fauces, or pharynx is frequently the primary point of invasion.

The human subject is also occasionally infected by ingestion, but such cases are apparently very rare in this country. That is not entirely, probably not even mainly, due to the fact that the flesh of anthrax animals is rarely consumed with us, but because anthrax flesh generally contains only bacilli (not spores), and these are destroyed during the process of cooking.

Susceptibility.—All the common domestic mammals are susceptible to anthrax. In this country the disease is most frequently diagnosed in animals of the ox species, and only a small number of cases in sheep figure in the official returns. It is not improbable that these returns are less trustworthy for sheep than for cattle as an indication of the prevalence of the disease, owing to the fact that farmers are more careless regarding the cause of death in the former species than in the latter, and also because there is a greater probability of the nature of the disease being overlooked in sheep if the blood is not examined microscopically. At the same time, it is not possible to believe that anthrax is a common ovine disease in Great Britain. In the human subject cases of malignant pustule contracted while dressing the carcases of sheep appear to be unknown in this country, and that would certainly not be the case if anthrax were a common disease among sheep. The greater frequency of the disease among cattle is not very easy to explain, except on the supposition that they are more susceptible than sheep to alimentary infection. It is well known that sheep are much more susceptible to experimental inoculation than cattle, the disease when thus set up with a strong virus being almost invariably fatal in the former species, while a considerable proportion of cattle recover from inoculation anthrax.

In point of susceptibility the horse appears to stand near the ox.

The pig is readily infected with anthrax by ingestion, although it appears to be more resistant to inoculation than cattle or sheep. The dog is generally credited with a marked insusceptibility to the disease, and it has even been by some authors declared immune. In this country anthrax has frequently been observed in the dog as the result

of feeding with raw anthrax flesh, and old as well as young animals have been attacked.

The domestic fowl, it is generally stated, is quite immune against anthrax in ordinary conditions, but it may be rendered susceptible by circumstances that lower the vitality, such as immersion in cold water until the temperature is reduced several degrees.

The mouse, rabbit, and guinea-pig are very susceptible to anthrax by inoculation, but they cannot with anything like the same certainty be infected by feeding.

Neither age nor general condition appears to affect susceptibility, and no difference in the degree of resistance to infection has been noted in different breeds of the same species in this country, though Chauveau found that Algerian sheep possess a high degree of natural immunity against anthrax.

Symptoms.—In a very large proportion of cases of anthrax in the ox and sheep the period during which manifest symptoms of illness are exhibited does not exceed a few hours, and it therefore frequently happens that animals thought to be perfectly healthy when last seen are found dead. Systematic employment of the thermometer in out-breaks of the disease shows that the earliest indication of infection is a rise of temperature. This will often be found to be from 2° to 4° above the normal in animals that are still eating and ruminating. Moreover, although it is impossible to doubt that this febrile state is really a mark of infection, many animals that have for a day or two a temperature decidedly above the normal recover without developing any urgent symptom.

Clinical observation thus shows that anthrax in the ox is by no means so invariably fatal as many suppose it to be, and this view is also borne out by experiment, since a considerable proportion of cattle inoculated with virulent anthrax blood or culture recover.

The elevation of internal temperature may be associated with slight dulness, inequality of surface temperature, impairment of appetite, occasional rigors, and acceleration of the heart's action, and these also may pass off in a day or two.

In the fatal cases the urgent symptoms rapidly follow on one another, and the animal generally dies within an hour or two of their onset. These are, great prostration, profound dulness of expression, rapid action of the heart, and feeble pulsation in the arteries. Occasionally more or less blood is passed with the fæces. Death usually takes place quietly, without convulsions.

In the sheep the symptoms are very similar, but the course of the disease is even more rapid, the period during which urgent symptoms are exhibited rarely exceeding an hour or two. During this period there are signs of intense prostration, and both urine and fæces may be tinged with blood. As in the case of the ox, death usually occurs without convulsions.

In the horse a somewhat longer period, from four to twelve hours, generally elapses between the onset of pronounced symptoms and death. Indications of abdominal pain are in many cases exhibited, the animal being more or less restless, lying down and rising up again frequently, and perspiring. The temperature rises 3° or 4°, the respirations are hurried, and the heart's action is rapid. In a con-siderable number of cases, the signs of colic are slight or absent, but

a new symptom is added in the shape of a diffuse swelling in the region of the throat. This is often the earliest abnormality noticed. The swelling is hot, tense, and ill-defined at its margins, rapidly spreads down the neck, and may almost reach the entrance to the chest before death takes place. With this localisation of the disease, more or less urgent dyspnœa is one of the most striking symptoms.

In very rare instances the disease is ushered in by the sudden development of a brawny swelling in some other part of the horse's body, and occasionally in an animal that has displayed the earlier symptoms, such as rise of temperature, general depression, loss of appetite, etc., a number of such swellings may form in succession in different places, and ultimately disperse, the animal recovering after an illness extending over some days.

In the pig primary localisation of the disease in the throat is the rule, and it is also frequently observed in the dog. The more or less urgent dyspnœa caused by the swelling of the throat and neck is accompanied by the general symptoms seen in other animals—elevation of temperature, rapid action of the heart, rigors, complete loss of appetite, and great prostration. The duration of the illness lasts from a few hours to a day.

Lesions of Anthrax.—In an animal dead of anthrax putrefactive changes set in immediately, and proceed with great rapidity. Within a very short time after death the stomach and intestines become enormously distended with gas, in consequence of which the anus becomes everted, and the abdominal parietes occasionally ruptured. A blood-stained fluid escapes from the anus, and a similarly tinged froth is expelled from the nostrils. In sheep the skin soon shows a bluish-black discoloration, and the wool is easily pulled out. This proneness to rapid putrefaction is in no way characteristic of anthrax. It is simply due to the deoxygenated state of the blood at the moment of death, this being eminently favourable to the multiplication of the malignant œdema bacillus and other putrefactive anærobes.

The subcutaneous veins are distended with dark imperfectly coagulated blood. If this blood be examined before putrefaction has made progress, it will be found that the white corpuscles are abnormally numerous (hyperleucocytosis). Extravasations of blood may be present under the skin, but except at the seat of local swellings, such as that of the throat, these are generally absent. Within the abdomen the most constant lesions involve the spleen, the stomach (fourth stomach in ruminants), the intestines, and the lymphatic glands.

Engorgement of the spleen is almost invariably present in anthrax in cattle (hence the term splenic apoplexy). In the great majority of cases that organ is from five to ten times the normal volume and weight, and sometimes the congestion is so great as to cause rupture of the splenic capsule. The spleen pulp is softened, often diffluent and dark in colour, though it may brighten on exposure to the air. In rare cases of bovine anthrax, however, the spleen may be normal in size, and almost normal in appearance, and exceptions to the rule are commoner in the other domestic animals. In the sheep particularly, the spleen, although more or less softened, is often normal in size, or but slightly enlarged. The stomach or intestines, or both, are

frequently inflamed, the inflammation being of a hæmorrhagic type. The intestinal contents may be mixed with more or less liquid blood. The enteritis sometimes affects the small intestines mainly, at other times the colon and cæcum are mainly involved. In the one case the mesenteric glands, and in the other the lymphatic glands of the large intestine, are swollen, deeply congested, and hæmorrhagic. The liver and kidneys as a rule are normal save that they are congested. The organs of the thoracic cavity are generally normal, but the pleura, the serous membrane of the pericardium, or the endocardium may show hæmorrhagic spots.

In those cases in which swelling of the throat is a symptom during life the subcutaneous and intermuscular tissue of the region is found to be saturated with a straw-coloured, clear, trembling, gelatinous exudate, and this may be associated with larger or smaller blood extravasations. The lymphatic glands of the throat are then swollen, congested, and hæmorrhagic. In those rarer natural cases in which circumscribed swellings are present in other parts of the body, and at the seat of inoculation in experimental cases, the swelling is found to be due to the presence of the same straw-coloured gelatinous exudate. The transparent character of this exudate is due to its poverty in leucocytes, and that again is referable to the negative chemotaxis which anthrax bacilli and their products exert on the white blood corpuscles.

Diagnosis during Life.—The diagnosis of anthrax during life is in certain circumstances comparatively easy and in others attended with great difficulty. It is easy when, as is the rule in the pig, both the history and the external manifestations point to anthrax. The writer has had brought under his notice but one outbreak of anthrax in the pig in which there was not a history of the animals having recently been fed either with slaughter-house offal, or with the raw flesh of some farm animal found dead or which had died after a very brief illness. When with such a history some of the affected pigs show swelling of the throat and neck anthrax may confidently be diagnosed. In cattle swelling of the throat or other external manifestations of anthrax are rarely present, but when there is a history of recent sudden death of one or more animals, and some of other members of the herd are found to have an elevated temperature, a probable diagnosis may be made. In the horse, unless there is swelling of the throat, the first case is not likely to be correctly diagnosed owing to the comparative rarity of the disease among horses in this country, and the difficulty in distinguishing between the symptoms displayed and those of enteritis, peritonitis, or twist of the bowel. It is unnecessary to discuss the *intra-vitam* diagnosis of anthrax in the sheep, as owing to the extremely rapid course of the disease in members of that species the veterinary surgeon rarely has the opportunity to see the patient while alive.

Diagnosis after Death.—In the great majority of cases, whatever be the species attacked, the veterinary surgeon has a dead animal at his disposal when he is first called in to an outbreak of anthrax, and in that case a perfectly certain diagnosis may quickly be made by anyone who is acquainted with the characters of the anthrax bacillus as it is found in the blood. But, in this matter there are several pitfalls into which the tyro is very likely to drop. Probably the most

dangerous of these is the resemblance between anthrax bacilli and certain putrefactive organisms, particularly the bacillus of malignant œdema, when insufficient powers of the microscope are used. This source of error is absent in the perfectly fresh carcase, and except during the warm months of the year it is also absent for twenty-four hours or more after death if the blood for examination be taken from one of the veins of an ear or a foot, as the invasion of the blood by putrefactive organisms has not then reached the extremities. The practitioner who aspires to basing his diagnosis of anthrax on microscopic examination of the blood, must before he attempts to put his knowledge into practice have satisfied himself that he can certainly distinguish between a preparation of fresh anthrax blood and one of putrid blood containing large numbers of malignant œdema bacilli. It would be a great mistake to suppose that this is easy, and it is not improbable that fully as many errors are at the present time made by those who base their diagnosis on the result of a microscopic examination as by those who rely solely on the history and the appearance of the lesions.

For simple diagnostic purposes aqueous solution of methylene blue is probably the best stain in the hands of those who are not expert bacteriologists. A convenient solution may be made by dissolving 1 part of methylene blue in 100 parts of ½ per cent. carbolised water, filtering, and preserving in a glass-stoppered bottle. Such a solution will keep for an indefinite period. One advantage of this solution is that it stains the bacilli of malignant œdema much more intensely than the anthrax bacilli. In blood that is partially putrid the anthrax bacilli, provided they have not entirely disappeared, are by this method stained of a light blue, which is in strong contrast with the blue-black colour of the associated putrefactive organisms. Indeed, when intensely staining bacilli are found in blood that is not quite fresh it may safely be concluded that they are not anthrax bacilli.

The trouble necessary to enable one to certainly distinguish between anthrax bacilli and putrefactive bacteria is well spent, for microscopic examination of the blood is the only method that entirely obviates the necessity of opening the body in order to arrive at a diagnosis.

With a sufficient magnification anthrax bacilli may readily be detected in unstained preparations of blood, and some authors have even recommended this method of examination as preferable to the use of stained preparations. It has the obvious merit of being simpler, but in inexperienced hands it is certainly less reliable.

In all circumstances the blood for microscopic examination should be taken from one of the peripheral veins, the ear being the most convenient part. This course is commendable for a double reason. In the first place, it reduces to a minimum the amount of blood placed in contact with the air, and this, as already explained, is in the highest degree desirable. In the second place, in partially putrid carcases anthrax bacilli may be found in the blood of the peripheral veins a considerable time after they have entirely disappeared from the veins of the great body cavities.

The experimental inoculation of small animals is not of great value in the diagnosis of anthrax, and not infrequently it conducts those who adopt it into absolute error. When the suspected carcase is still

fresh inoculation of a guinea-pig or rabbit with a trace of blood justifies the diagnosis "not anthrax" if the result of the experiment is negative, but death of the experimental animal by no means justifies an opposite conclusion. Before arriving at that opinion it would be necessary to find anthrax bacilli in the blood of the guinea-pig or rabbit, and whoever is capable of doing that might have found the bacilli in the same way in the blood of the suspected carcase.

When the suspected carcase is partially putrid, inoculation experiments uncontrolled by microscopic examination of the blood of the experimental animal are in the highest degree fallacious, because many putrefactive bacteria, including the frequently present malignant œdema bacillus, are highly pathogenic for the guinea-pig and the rabbit. A further source of error is introduced by the fact that putrefaction destroys anthrax bacilli, and inoculation with such materials as putrid spleen pulp may fail to communicate anthrax, although the case was really of that nature.

Inoculation experiments may usefully be resorted to when, as in partially putrid blood, anthrax bacilli cannot with certainty be identified by microscopic examination, but when such an experiment entails the death of the experimental animal the blood of the latter must be microscopically examined before deciding that the case is one of anthrax. Moreover, a diagnosis is justified only when the experiment has a positive result, for partially putrid anthrax blood may fail to infect owing to all its contained bacilli having perished.

When experimental inoculation is resorted to the guinea-pig ought to be preferred to the rabbit, unless the blood is perfectly fresh, as the former is almost immune against certain putrefactive bacteria that are intensely virulent for the rabbit. When partially putrid blood or spleen pulp is used it is also better to rub the suspected material into the scarified skin than to inject it hypodermically, as the malignant œdema bacillus, although readily fatal by subcutaneous injection, generally fails to infect by endermic inoculation, while the latter method is quite reliable as a means of infecting with anthrax.

The practitioner who cannot rely on his own microscopic examination as a guide to diagnosis in suspected cases has several courses open to him. (1) He may cut off an ear or a foot, send it to some competent bacteriologist, and in the meantime take all the precautions necessary in a case of anthrax. (2) He may base his diagnosis on the clinical history and the external appearance of the carcase, and abstain from a proper *post-mortem* examination. (3) He may proceed to open the body in order to base his opinion on the condition of the internal organs. The first of these three courses is undoubtedly the best, and the last is probably the worst, since it is dangerous to the person operating and to those who assist him, and involves serious contamination of the ground, with the greatly to be dreaded danger of subsequent spore formation. The opening of the carcase of an animal supposed to have died of anthrax ought to be forbidden by law until a microscopic examination by some competent person has proved that the disease was of another nature.

Assuming, however, that the practitioner has decided to open the carcase, it becomes necessary to discuss the value of the indications which he may thus obtain. In the case of cattle, if the carcase is moderately fresh, a tolerably well-founded, but not absolutely certain,

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opinion may be based on the condition of the spleen. If the animal has been unexpectedly found dead, or has died after a few hours illness, and the spleen is found to be decidedly enlarged and softened, there is little chance of error in diagnosing the case as one of anthrax. The diagnosis cannot be absolutely certain because (1) in a very small proportion of cases in cattle the spleen is almost normal in appearance, and (2) because extreme splenic congestion is occasionally found in other conditions than anthrax.

What has just been said of the ox applies, for the most part, to the horse also, though in the latter species exceptions to the apoplectic condition of the spleen are probably more numerous. But, on the other hand, swelling of the throat is much more frequent in the horse, and where the peculiar straw-coloured gelatinous exudate is found in that region, and there is enteritis, with great enlargement and congestion of the intestinal lymphatic glands, anthrax may be diagnosed with little chance of error. In the pig and dog also these lesions may confidently be set down to anthrax, especially when, as is nearly always the case, there is a history of the animal having recently been fed with raw flesh.

Diagnosis based solely on *post-mortem* appearances is least satisfactory in the case of the sheep, because in that species swelling of the spleen is far from constant, and because the lesions in other parts of the body are generally seriously masked by the putrefactive changes that have taken place before the carcase is submitted for examination.

Treatment.—Notwithstanding a contrary belief entertained by a good many veterinary surgeons, it may confidently be asserted that the course of an attack of anthrax remains entirely unaffected by any medicinal treatment at our command. The belief that such agents as carbolic acid or hyposulphite of soda, administered by the mouth, exercise a curative affect on anthrax is an erroneous one, arrived at apparently because many have not recognised that the forces of nature are sufficient to bring a considerable proportion of the larger animals successfully through an attack of the disease.

Prevention.—The measures that may be taken with the object of prevention comprise (1) protective inoculation, or vaccination, and (2) those precautions against infection which are suggested by a knowledge of the methods by which the disease is generally contracted and spread.

The Pasteurian method of vaccination has had a most extensive trial in France and several other countries in which anthrax is specially prevalent, and it is claimed for it that it has effected a marked reduction in the death-rate from the disease in districts in which it has been systematically practised. The classical experiment which was carried out at Pouilly-le-Fort in 1881 proved in the most convincing manner that the Pasteurian method of vaccination, when properly performed, confers a high degree of immunity against anthrax— an immunity which enables vaccinated sheep to resist inoculation with quantities of virus certainly fatal to ordinary unvaccinated animals of the same species. Nevertheless the method has not found universal favour, and in this country it has had a very restricted trial. Probably one of the reasons why it has not been largely practised with us is that anthrax in this country is seldom such a formidable plague as it is in many other countries. Evidence of this is afforded by the fact that

the total number of animals returned in any one year as having suffered from anthrax has only once exceeded 1000 (1300 in 1893), while the average number of fatal cases in an outbreak is generally under two. Another obstacle to the general adoption of vaccination by British stock - owners, has doubtless been the large proportion of accidents that have attended the operation in some of the few instances in which it has been resorted to in this country. A notable example of such untoward consequences of vaccination was recorded by the writer in Vol. VII. of this Journal (p. 325). In that instance out of a lot of 225 ewes sixteen died as the result of the second vaccination. A still greater shock to one's faith in the usefulness of vaccination was imparted by the fact that when, less than four months afterwards, a number of the vaccinated ewes of this flock had their immunity tested by feeding or inoculation with strong anthrax virus, they all succumbed to the disease. This case, and others of the same kind that have occurred abroad, show that the preparation of an anthrax vaccin that is at once safe and efficacious is not a matter that can be regulated with mathematical accuracy, and suggest that it would be very unwise for any British veterinary surgeon to press this method of dealing with the disease on a reluctant stock-owner.

Other methods of protective inoculation, by processes of chemical vaccination, may be dismissed by saying that while some experiments on a laboratory scale encourage the hope that we may yet be provided with a germ-free vaccin that will confer a valuable degree of protection, we do not yet know any method of that sort which we can recommend to the farmer. In this connection it is interesting to recall that Pasteur himself, before he had discovered the method of vaccinating by attenuated cultures, expressed his firm conviction that anthrax could easily be exterminated by exercising proper care in disposing of animals dead of the disease.

In considering what prophylactic measures other than vaccination may be taken when an outbreak of anthrax occurs, it is necessary to remember that every animal affected with the disease is a source of soil contamination while it is still alive, by reason of the bacilli voided in its urine or excrement, and that it may be made a still more serious source of contamination after death, if its carcase is improperly dealt with. To limit the dangers arising in the first of these ways every animal suspected of anthrax (and, as previously mentioned, when an outbreak occurs many cases may be diagnosed early by the use of the thermometer) ought if possible to be confined in a place that admits of being thoroughly disinfected. The dung, bedding, etc., in the place should afterwards be destroyed by burning, and the floor and walls ought to be treated with some reliable germicide. If the disinfection is practised immediately, and the temperature does not exceed 70° F., it may safely be considered that any disinfectant capable of destroying spore-free bacilli will suffice, such as a 2 or 3 per cent. solution of carbolic acid, creolin, or chloride of lime. On the other hand, if the temperature has been 80° and a day or more has elapsed since the contamination occurred, rendering it probable that spores may have been formed, stronger agents, such as $\frac{1}{2}$ per cent. mercuric chloride, ought to be employed. It is also worth remembering that the disinfectant power of any of these solutions is intensified by using them hot.

Every case of sudden unexpected death in cattle, sheep, or horses ought to be suspected of anthrax until the contrary is ascertained. If this precaution were invariably adopted the lives of human beings as well as of the lower animals would occasionally be saved. When the case is ascertained to be one of anthrax the method of disposing of its carcase will depend upon the place where the death has occurred. If the carcase is found in a field, and the soil admits, a grave of sufficient depth (not less than six feet according to the regulations of the Board of Agriculture) should be made alongside the carcase, and in this the latter and any visibly contaminated earth in its neighbourhood should be buried. If the burial is promptly effected, even in summer time, putrefaction and the deprivation of oxygen may be relied upon to destroy the bacilli before they have had opportunity to resolve themselves into spores. As a further precaution, some reliable disinfectant, such as chloride of lime, may be applied to the surface of the soil where the carcase lay, or a thick layer of litter may be spread on the place and then set fire to.

If the death has occurred in a building, or in a field or other place not adapted for the making of a grave, the carcase must be transported to some place where it can be destroyed or buried in the manner above described. During this transport adequate precautions to prevent dissemination of infective material from the natural orifices of the body ought to be taken, and the vehicle used to convey the carcase should afterwards be thoroughly cleansed and disinfected.

As a precaution intended to prevent the transmission of the disease to the pig and dog, farmers ought to be warned against the danger of feeding either of these animals with the raw flesh of any farm animal that has suddenly died from some unknown cause.

Finally, when an outbreak of anthrax occurs among animals at grass, it is advisable to keep animals off the field in which the death occurred as long as possible, in the hope that such germs as must almost inevitably have been deposited on the surface of the soil or pasture may either be washed away by rain, or be destroyed by desiccation, sunlight, or the competition with more rapidly growing soil organisms.

DESCRIPTION OF PLATE I.

Fɪɢ. 1.   Anthrax bacilli from spleen of rabbit, stained with methylene blue, mounted in water (× 535).

Fɪɢ. 2.   Anthrax bacilli from spleen of mouse, stained with gentian violet, washed in alcoholic solution of picric acid, mounted in Canada balsam (× 1000).   The envelope of the bacilli is unstained and invisible.

Fɪɢs. 3. and 4.   Anthrax bacilli from same source as in Fig. 2, stained with gentian violet, washed with acetic acid, mounted in water (× 1000).   In Fig. 3 the envelope of the bacilli is distinctly visible.

Fɪɢ. 5.   Sporulating anthrax filaments from an agar culture, stained with carbol-fuchsin (× 535).   The envelope is well shown.

Fɪɢ. 6.   An anthrax colony from a gelatine plate cultivation (× 30).

Fig. 1

Fig. 2

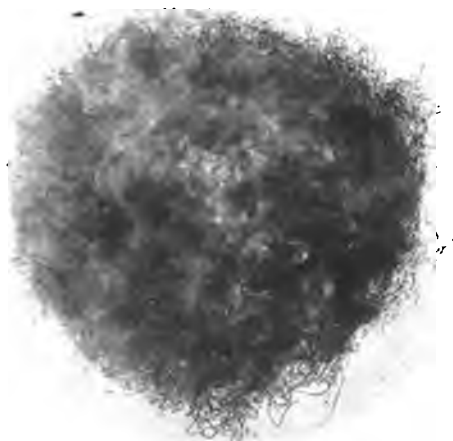
Fig. 3

Fig. 4

Fig. 5

Fig. 6

OBSERVATIONS ON SURGICAL CASES.

By JAMES MACQUEEN, F.R.C.V.S., Royal Veterinary College,
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DEFECTIVE teeth.—Imperfect mastication and quidding in horses,
when not due to wound, abscess, foreign body such as wire, nail, or
wood lodged in the mouth, or to tetanus, is caused generally by some
defect of the molar teeth. Sharp edged, irregular, prominent, or "over-
grown" molars are common enough, but carious teeth are compara-
tively rare. The causation of defective teeth is perhaps less important
than interesting, but it is usual to ascribe irregularities of the molar
tables to unequal development of the jaws. Faulty development may
not satisfactorily explain the occurrence on the grinding surfaces of a
series of responding pits and projections an inch or more in depth or
height, but it supplies a convenient answer to a difficult question.
Other explanations have been given, but at best they are speculative
and unconvincing.

In some horses, owing probably to unequal length of the jaws,
the incisor tables do not respond exactly to each other. Cases
of overshot or undershot incisors, though varying greatly in degree,
are nearly always associated with the formation of prominences
more or less large on the first and sixth molar teeth. In overshot
mouth a portion of the tables of the first upper and sixth lower
molars escapes wear and projects, and in undershot mouth
the unworn part occurs on the first lower and sixth upper
molars. The prominences of the first molars seldom cause incon-
venience, but their existence in any case of slow or imperfect masti-
cation ought to suggest careful examination of the other teeth.
Unworn or protuberant parts of the sixth molars of the lower jaw
may be found sloping backwards or inwards, or standing upright and
partly imbedded in the palate or gum.

At first the molar tables are fairly level; later, in consequence of
wear and, to some extent, of the form of the surfaces of the maxillary
joints, the tables become oblique. One effect of the inclination of the
grinding surfaces is to sharpen the inner edge of the lower and the
outer edge of the upper teeth. If the lower molar arch is too narrow,
or the upper arch too wide, inclination of the tables increases, and in
time the crowns cross like shears blades. Shear teeth usually preserve
their full length, and it is customary to refer to them incorrectly as
"overgrown." In the horse gradual elevation of the molar teeth is a
natural process. As wear advances at the crown the implanted
portion loses volume, and the alveolar cavity shrinks as the tooth
ascends. Shear teeth have been less worn at their tables than other
teeth, but they are not overgrown. After extraction of a molar the
tooth opposed to the gap in the table is no longer worn or restrained
by direct pressure. It rises in its socket a little faster than the adjoin-
ing teeth, but it does not increase in bulk. If not cut or extracted
this tooth eventually bruises the gum or palate, or enters the vacant
alveolus and impedes mastication.

Frequently in aged cart horses, and occasionally in horses under
five years, imperfect mastication or quidding, with emaciation, may be
traced to the formation of spaces between the crowns of the molar

teeth.   Food particles fill the spaces, and sink between the teeth and gum.   Caries may attack the crown, but oftener some product of bacterial action in the retained food irritates the alveolar lining, causing periostitis and abscess.   Some of the most intractable dental cases have been connected with the lodgment of food between the molar crowns.   The disease process advances slowly, and early extraction of the loosened teeth may prevent farther extension.   In neglected cases, or in those complicated by abscess and necrosis, the jaw enlarges, pus tracts run from the suppurating alveolus through the expanded bone to a secondary or gravitation abscess, which forms in the submaxillary space, and alveolar fistula may prevent recovery. It should be stated that although the teeth in these cases are loose, extraction may be difficult in consequence of enlargement of the fangs by tuberous growths.

As a preliminary to treatment the patient should be carefully examined, and examination should not be restricted to the teeth. This warning cannot be too rigidly observed when dealing with old horses, in which defects of teeth may co-exist with a disease which makes slaughter preferable to any operation.   In dental as in other cases prevention should be better than cure, but preventive treatment is generally impracticable when not impossible. The horse's molars are not often amenable to the art of hygiene which occasionally prescribes measures that cannot be successfully applied. Curative treatment, if less worthy, is more popular than preventive treatment, and, in tooth troubles, far more efficacious.   Dentists— equine and canine—are gaining the "Crest of the wave," which sooner or later must wash them into remunerative havens.   But they should see to their appliances, which, too often, are both cumbersome and costly. Unlike scalpels and other instruments of a nicer sort, tools for teeth do not readily wear out ; and it is not improbable that a few of the heavier variety now used have been handed down from one generation to another.   Tooth instruments of alarming size—too large for convenient use—are far too common, but, with no pretence to prophesy, the hope may be entertained that the day is not distant when old-fashioned dental implements will be allowed to rust in their cases, and files of various shapes, hack saws, and circular saws mounted on arms like those of the dentist's drilling machine, will displace the clumsy rasps and clumsier shears.   Then, instead of extracting carious teeth, which,· by the way, are often fairly sound, cavities will be stopped with the best amalgam.   But too much must not be expected from posterity.   We must hasten slowly, and at least for a time content ourselves with the tools we have got.   Sharp molar edges can be rounded with the rasp, and small projections can be removed by the chisel or by the odontriteur.   For prominences of the last molars the chisel can be used, but not without risk to the gum or palate, and if the protuberance is thick the force employed may displace the tooth.   Failure to cut, at the first blow, a projection from the sixth upper molar may result in fracture of the alveolar wall and sinking of the tooth into the maxillary sinus.   When a similar accident happens in attempting to cut the last lower molar, the result is less serious, probably because the displaced tooth remains concealed. Tooth shears cannot always be used successfully in these cases. Sometimes, owing to insufficient space or to the form or direction of

the prominence, the shears cannot be set across its length. By chance rather than by skilful guidance the protuberance may be cut, but oftener the shears slip off after merely pinching the prominence, which may be too narrow for space between the closed blades. Occasionally perseverance has been rewarded by oblique fracture extending to the fang, and although the offending projection was replaced by a pit it is doubtful which condition gave more trouble. For shortening teeth that protrude beyond the level of the table, and for cutting shear teeth, Thompson's shears are very good, though sometimes a tooth is loosened as well as cut and extraction must follow sooner or later. Fracture in a bad direction and displacement, accidents which occasionally qualify the action of shears on a projecting tooth, can be avoided by employing a saw. One of the best for the purpose and the least expensive is the American hack-saw extended in a narrow frame. When using this saw, which is very brittle, care should be taken not to jerk or bend it, and to prevent abrasion of the cheek a smooth-edged plate of zinc should be inserted between the cheek and teeth. How far the hack-saw will be able to assist operators on protuberant teeth cannot be stated positively before further trial. It will cut a molar in a few minutes, but it cannot be used on the sixth molar because of insufficient space behind the tooth to work the saw.

Attempts have been made to save carious molars by scraping and cleansing, and afterwards filling the cavities with various tooth substitutes, on lines similar to those pursued by dentists in the treatment of human teeth, but the relief given lacked permanency. The dentist's drill was not thought of; had it been used in preparing the cavities for the stopping, the result might have justified continuance of a branch of practice which has been too much neglected by veterinary surgeons. When extraction must be performed, forceps should be tried in all cases where the crown of the tooth can be securely grasped. Santy's forceps will draw any ordinary molar which is not chisel-crowned or brittle from the ravages of caries. The fulcrum supplied with the forceps concentrates too much pressure on a single tooth, which may subside during operation. A short rasp or chisel forms a safer fulcrum. In operating strength is useful if not all important, and art is helpful, but the main thing is the application of the forceps to the tooth—the grasp must be secure. Teeth which have been crushed or fractured at the crown by forceps, and some carious teeth, can only be removed by expulsion or punching out. The upper molars can be reached without difficulty in old horses by trephining the wall of the facial sinus ; but in young horses the root of the sixth molar lies too near the orbital case to permit of safe punching. When this tooth must be thrust out a bent punch should be used. The first five lower molars can be punched out after trephining the horizontal border of the maxilla, but the sixth lower tooth, owing to varying direction or the distance of its root from the border, cannot be expelled without risk of damage to the jaw.

Compared with extraction by forceps, expulsion is less difficult, especially in old horses, and decidedly more painful in either old or young. Besides, the healing process is very slow and seldom satisfactory. The opening necessary to operation on the lower teeth can be cleansed and drained, and, perhaps, healed within reasonable time, but the breach made by the punch in the wall of

an upper tooth socket exposes the facial sinus to infection, and it must be confessed that contamination very frequently occurs. Food gains access to the sinus, and, until the alveolus becomes closed, the first trephine hole, or a second, which may have been made for better drainage, must be kept open. When two upper molars have been punched out the probability of complete recovery is exceedingly doubtful. The gap in the gum diminishes, but the opening leading from the sinus into the alveolus remains in spite of daily attention. In some cases, after constant treatment extending over many months, a medium-sized probe may be passed readily through the partly filled alveolus from the mouth to the sinus. Veterinary surgeons, of more or less experience, sometimes regard the expulsion of molar teeth as a mere surgical bagatelle, and the subsequent treatment as of little consequence. But reports of rapid and complete healing after the punching operation should only be accepted with respectful reserve. Extraction by forceps of an upper molar leaves the bony wall of the alveolus intact, but, in punching, the roof or cap of the alveolus is partly removed. In the first instance food may, and generally does, accumulate in the empty socket without appreciable inconvenience to the horse, and in the second some of the food gains the interior of the sinus. To shut off communication between mouth and sinus the alveolus must be closed at the gum, at the sinus, or between these limits. To expect rapid closure is absurd, and to expect perfect obliteration of the passage, before a very long while, is somewhat akin to the expectation of the man who sought to convert a tin-can into a solid by repeatedly coating its inside with paint. Enthusiasm "is cheap to-day," indeed, it bids fair to supersede the truth. Equine dental surgery has its disabilities, and to indicate some of these is the purport of the foregoing observations.

EDITORIAL ARTICLES.

THE STAMPING OUT OF RABIES.

THE recent marked reduction in the number of cases of rabies must be disappointing to the numerous false prophets who foretold that the efforts of the Board of Agriculture to stamp out the disease would end in failure. It is true we have probably not yet seen the last case, but it is now impossible to doubt that a continuance of the existing regulations will before long be crowned with complete success. The number of cases of rabies in the dog reported in 1896 was 438, as against 154 in 1897, and the numbers for the first eleven weeks of 1897 and 1898 were respectively 44 and 6. It is well, however, to remember that this is not the first occasion on which rabies has been brought to the verge of extermination. In 1889, 312 cases of rabies in the dog were returned, and for the following three years the figures were 129, 79, and 38, respectively. This marked reduction in the number of cases was traceable to the enforcement of muzzling regulations in

those districts in which the disease was most prevalent, and the subsequent recrudescence of the malady, until in 1895, 672 cases were returned, was due to the laxity of Local Authorities in using the powers conferred on them by the then existing Rabies Order. There is, fortunately, little occasion to fear that that episode in the history of rabies will be repeated, for the large measure of success that has attended the present method of dealing with the disease has practically disarmed the opponents of muzzling.

There can be no doubt that the rapid decline in the number of cases since the latter part of 1897 is almost entirely ascribable to the consistent enforcement of the muzzle in every district in which a case of the disease has been discovered. It is true that some writers on rabies attach great importance to the destruction of so-called ownerless dogs as a means of checking the disease, and it has even been maintained that it is chiefly among such animals that the disease is propagated. Without denying that there may be countries in which rabies is mainly spread by the agency of ownerless dogs, it may with confidence be asserted that such is not the case in Great Britain, for the very good reason that the number of homeless dogs in this country is quite inconsiderable. A dog that has strayed from home may be said to be homeless, but there is no reason to think that such a dog is exposed to any extra risk of being bitten by a rabid dog. In short, the belief that we have in this country any considerable number of dogs unattached to human habitations, is entirely erroneous, and the wholesale destruction of so-called ownerless dogs is effectual against rabies only because the net that is spread to catch such animals must necessarily catch also the rabid dog, which, although seldom or never ownerless, very frequently strays from his home.

One of the main objects of repressive regulations must be to counteract this tendency on the part of rabid dogs to wander from home. The relative value of the collar and muzzle from this point of view has been much discussed. The experience of some foreign countries makes it impossible to deny that the compulsory use of a collar with the name of the owner engraved on it is an effectual means of checking the spread of rabies, but it is not at all certain that it would ever suffice to stamp out the disease from a country with such great numbers of dogs in small areas as are to be found in the large towns of Great Britain. That it probably would be very slow in its operation is shown by the experience of Bavaria, where, with the general use of a collar, a period of ten years did not suffice to eradicate the disease.

The superiority of the muzzle over the collar would appear to be incontestable on *a priori* grounds, and no argument drawn from experience points in the opposite direction. As an argument against the use of the muzzle it is often said that rabid dogs are never found with muzzles on them. The falsity of the statement has frequently

been exposed, but in advocating the superiority of the muzzle it is not necessary to maintain that it acts entirely by the restraint against biting which it imposes on a rabid dog. Rabid dogs do at times escape from their homes and begin to wander with a muzzle on, and provided the muzzle is an efficient one the dog will be harmless, but an escaped rabid dog is not a whit less dangerous because he is wearing a collar. The main use of the muzzle, however, is to serve as a mark that the dog is strayed or out of control, and in that respect it is obviously greatly superior to a collar.

A no less vexed point is the question of local as against general muzzling. Strange to say, the loudest advocates of universal muzzling are to be found among those who are at heart opposed to muzzling in any shape or form, which suggests the suspicion that these people hope, by bringing about a general muzzling order, to raise such an amount of opposition to the practice as will sweep away all the restraints at present imposed on dogs. The preference for a muzzling order applicable to the whole kingdom is often founded on the alleged absurdity of compelling dogs in such and such a place to wear muzzles, while at another place twenty yards distant the dogs are free to go unmuzzled. But it hardly needs to be pointed out that if local muzzling fulfils the object in view there is no absurdity in not making it general, and that as long as it is not general muzzled and unmuzzled areas must absolutely touch each other. The only point to be considered is whether the system of issuing local muzzling orders or that of enforcing muzzling all over the country is most likely to exterminate rabies with the minimum of annoyance to dog-owners, and in this matter we range ourselves on the side of the Board of Agriculture. If, at the present moment, a case of rabies were discovered in the centre of Sutherlandshire, it would surely be more absurd to enforce a muzzling order in the whole of Scotland than to issue one applicable within a radius of thirty miles of the place where the case of rabies occurred. The inconvenience, we will not say the cruelty, of muzzling has been greatly exaggerated, but no one will deny that the use of the muzzle is irksome both to the dog and his owner. Sensible people will put up with this temporary inconvenience, without demanding that it shall be needlessly imposed on other people more favourably circumstanced.

We think also that the Board of Agriculture acted wisely in giving exemption to packs of hounds and other sporting dogs. It is well known that such dogs have had no exemption from rabies in the past, but it is certain that animals of that class have done very little to spread rabies over the country. Doubtless a fox-hound or a greyhound when rabid and wandering is quite as dangerous as any other kind of dog, but the exemption from muzzling applies only when the animals are in the charge of competent persons.

The fact that the discovery of a case of rabies is now always

followed by an order imposing the use of the muzzle on all dogs in the locality makes the question of diagnosis even more important than it was before. When a veterinary surgeon has had an opportunity to observe the suspected animal throughout the course of its illness, a diagnosis approaching to certainty may be made, but, unfortunately, the diagnosis has in many cases to be based entirely on such indications as may be found in the dead body. It too frequently happens that the only history obtainable is that the dog was killed because he had bitten someone, and nothing reliable can be learned as to whether any of the characteristic symptoms of rabies were exhibited or not. In these circumstances the diagnosis must be based on the results of a *post-mortem* examination, or, better still, on those of experimental inoculation.

In our experience there is only one organ of the body whose state furnishes evidence of any value at the *post-mortem* examination of a dog suspected of rabies. That organ is the stomach. When that is healthy in appearance and filled with ordinary food materials, it may, with a confidence that is little short of certainty, be declared that the dog in question was not rabid. On the other hand, if the stomach is empty of ordinary food materials, but contains a notable quantity of such foreign substances as hay, straw, wood-shavings, etc., in the case of a dog that has bitten anyone, or displayed any of the characteristic symptoms of rabies, there remains very little room for doubt that the animal was rabid.

Circumstances connected with one of the cases referred to the Research Laboratory at the Royal Veterinary College during the past year are interesting in this connection. In this case the dog was killed after he had made unprovoked attacks on several people and bitten them. The dog was a stranger to the district, and his previous history could not be traced. The carcase was sent to the Royal Veterinary College, and the *post-mortem* revealed a condition which appeared to be opposed to the conclusion that the dog was mad, viz., the presence of a considerable quantity of animal food, mixed with some hair, in an apparently healthy stomach. Notwithstanding this, in view of the history that the dog had bitten several people without provocation, it was at once reported that the case was almost certainly one of rabies. This opinion was verified by experimental inoculations, and subsequent inquiry removed the apparent conflict between the clinical history and the condition of the stomach. It was ascertained that on the day on which the animal was killed, it had wandered into a machinery shed, and been seen by one of the workmen to eat a quantity of grease for lubricating machinery. At the *post-mortem* examination this had been mistaken for animal fat.

With the foregoing exception, in every case of rabies that has come under notice at the Royal Veterinary College within recent years, the stomach was empty save for a little brownish liquid

(saliva and mucus) or foreign bodies of the kind already mentioned.

When the stomach contains neither food materials nor foreign substances, and there is no clinical history to fall back upon, there remains only one way by which diagnosis can be made, viz., by resorting to the experimental inoculation of animals. The rabbit is the animal generally selected for such experiments, and the material used to inoculate is taken from the brain of the suspected dog. This material is injected under the coverings of the rabbit's brain, and when it is taken from the fresh brain of a rabid dog it leads with great certainty to the development of the same disease in the inoculated rabbit.

During the year 1897 thirty-one cases in which rabies was suspected were referred to the Royal Veterinary College for experimental investigation. In twenty-nine cases the animal suspected was a dog in one case it was a cow, and in another a sheep. In seventeen of the cases the experiments proved that the suspected animal had been the subject of rabies (fifteen dogs, one cow, and one sheep); in thirteen cases they showed that the animal had not been rabid, and in one case the experiments did not warrant a positive diagnosis.

It has been asked whether the inoculation test is a perfectly reliable one, and this may be answered by saying that when the brain to be tested is fresh, and a sufficient number of experimental animals are employed (at least three), the results enable one to say positively whether the case was one of rabies or not. Even when the brain is partially putrid a positive opinion may be justified by the experiments, but the test may in such a case fail, owing to the death of the inoculated animals from septic inflammation of the brain before the rabic virus has had time to develop. Apart from that source of failure, experiments made with quite putrid brains are not absolutely reliable, for putrefaction weakens the rabic virus and ultimately destroys it. In the case referred to above, in which the experiments left the nature of the case in doubt, the brain was very putrid, and five out of six rabbits inoculated from it died within a few days from septic inflammation of the coverings of the brain. The sixth rabbit survived, and did not subsequently develop any symptom of rabies. Nevertheless, it was not considered safe to report the case as certainly not rabies.

THE RECRUDESCENCE OF SWINE-FEVER.

IF credit is due to the Board of Agriculture for the vigour and success of their operations against rabies, the same cannot be said of their dealings with swine-fever. For nearly four years and a half the Board has had a free hand in dealing with this disease, and the plague is still with us. Moreover, if one may judge from the success—or non-success—of the Board's most recent efforts, it appears probable that we shall have more rather than less of the disease in the future. It will be recollected that the Board assumed the powers vested in them under the Act of 1893, with respect to swine-fever, on the 1st November 1893, and it may therefore be of interest to quote the official returns relating to the disease since the beginning of 1894. They are as follows:—

Year.	Outbreaks.	Swine Slaughtered as Diseased or Suspected.
1894	5682	56,296
1895	6305	69,931
1896	5166	79,586
1897	2155	40,432

These figures are not entirely unsatisfactory, for during the past year the number of outbreaks had fallen to less than half those of 1894. It will be observed, however, that the whole efforts of the Board of Agriculture during 1894 and 1895, which involved the slaughter of over 120,000 pigs, not to speak of the immense expense incurred in carrying out the duties of inspection, were entirely wasted so far as making any impression on the prevalence of the disease was concerned. It was not until the latter half of 1896 that any notable improvement in the state of affairs was effected, and, without doubt, that improvement was due to the restrictions on the movement of swine imposed by the Infected Areas Order, which came into force on the 21st of April 1896, and the Suspected Zones Order, introduced in August of the same year. The steady enforcement of the provisions of these orders in the localities in which outbreaks had been discovered soon began to tell upon the disease, and until the end of 1897 the returns for each month showed a decreasing number of cases. That eminently hopeful state of affairs came to an end at the beginning of the present year, as the following figures show:—

[TABLE.

Week Ending.	No. of Outbreaks.
18th December 1897	17
25th ,, ,,	20
1st January 1898	33
8th ,, ,,	38
15th ,, ,,	37
22nd ,, ,,	27
29th ,, ,,	49
5th February ,,	47
12th ,, ,,	56
19th ,, ,,	51
26th ,, ,,	44
5th March ,,	55
12th ,, ,,	45

A clue to the serious recrudescence of the disease disclosed by these figures may perhaps be found in the fact that, while in the month of February last outbreaks occurred in no fewer than thirty-six counties in England, Scotland, and Wales, the provisions of the Infected Areas Order were in force in only eleven districts, and those of the Suspected Zones Order in only four. This may be contrasted with the month of June last, when the number of outbreaks weekly was about the same as in February of this year, while the number of Infected Areas declared numbered nineteen, and the Suspected Zones nine.

It cannot be said that the public has displayed a want of patience in observing the results of the Board's operations against swine-fever. The difficulties of the task are fully apprehended, but it is impossible to avoid the conclusion that something is wrong when, within the space of two months, the average weekly outbreaks mount up from less than twenty to over fifty.

The Board will justly be held to have squandered an immense sum of money unless it succeeds in stamping out the disease in the near future, for the reduction of the outbreaks to fifty in the week would be an utterly inadequate recompense for the sacrifices that have already been made. The task is difficult, but if the same foresight in devising measures, and firmness in carrying them out, which the Board has displayed in dealing with rabies, had been employed against swine-fever, that disease would before this have been eradicated.

CONGENITAL TUBERCULOSIS.

DURING the latter part of 1896, through the medium of the *Veterinary Record*, a reward of one guinea was offered for each tuberculous new-born calf sent to the Research Laboratory at the Royal Veterinary College. This offer was made in consequence of a correspondence which was then being carried on in the columns of the before-mentioned Journal, and in which several veterinary surgeons, on the alleged ground of their own experience, strongly dissented from the view maintained by the Editor of this Journal to the effect that tuberculous disease is very rarely present in calves at birth, even when one or other parent is affected with that disease. In consequence of this offer only three calves supposed to be the subjects of congenital tuberculosis were forwarded to the laboratory during the succeeding twelve months, and only one of these was an example of the condition asked for. In one of the other two cases the lesions which were present in the liver had only a distant resemblance to true tubercles, and both by microscopic examination and by inoculation experiments their non-tuberculous nature was clearly established. In the remaining case the lesions were undoubtedly tuberculous, but the calf appeared to be at least five or six weeks old, and the stage of the disease did not make it at all certain that the calf had been infected prior to birth. This case is interesting as an illustration of the common tendency of those who favour the view that tuberculosis is generally, or at least frequently, inherited, to regard every case of the disease in young animals as an example of congenital tuberculosis. It hardly needs to be explained that a period of even a few weeks of extra-uterine life is quite sufficient to admit of the development of distinct lesions in a calf that was quite healthy when it came into the world.

As a matter of fact, the calf which was accepted as an example of congenital tuberculosis was killed five days after birth, but the lesions discovered in it were at a stage which made it impossible to doubt that they had been in existence before birth. This calf was forwarded by Mr Holburn, M.R.C.V.S., Manchester, and the fact that the cow which gave birth to it was killed a short time afterwards and submitted to *post-mortem* examination adds to the interest of the case. The cow in question was recognised to be in the last stages of tuberculosis before she calved, and after death she was found to be the subject of very widespread disease, involving, among other organs, the womb itself.

This case does not lend the least support to the view that in any notable proportion of cases tuberculosis is transmitted from the cow to the calf while the latter is still in the uterus, nor does it weaken the position of those who maintain that in the vast majority of cases a cow that is herself tuberculous will bear perfectly healthy calves. It has never been maintained by anyone that the intra-

uterine transmission of the disease never occurs, and it is generally admitted that when the uterus is the seat of tuberculous disease there is a great risk, though not even then a certainty, that the calf will be born tuberculous. But it is maintained that with a healthy uterus, no matter what the extent of the tuberculous disease is elsewhere, the progeny are probably never born tuberculous, and there is abundant experience to prove that the uterus is not one of the common seats of the disease in tuberculosis of the cow.

It has also been asserted by those who believe in the frequent occurrence of congenital tuberculosis that the testicles are not rarely the seat of tuberculous disease, and this statement is put forward as if it afforded an explanation of the alleged frequency of tuberculosis in the progeny of particular bulls. During the past year several testicles from rams and bulls were sent to the laboratory for examination, and one of these, forwarded by Mr A. Pottie, Jr., M.R.C.V.S., Paisley, was found to be the seat of genuine tuberculous disease, while the others were either healthy or showed other structural alterations easily distinguished from true tubercles. Again it may be said that the occurrence of tuberculous disease of the testicle of the bull has never been denied, but the condition is still rarer than tuberculosis of the womb, and on that account has even less bearing than the latter on the general question of congenital tuberculosis.

Finally, in connection with this subject, it may be pointed out that the question as to the relative frequency with which tuberculosis is transmitted from the mother to the unborn fœtus, and the influence which that ought to have on measures devised to prevent or eradicate the disease, has almost entirely lost its importance since the discovery of tuberculin. When a pregnant cow has reacted to tuberculin it is waste of time to discuss the probability of her calf being tuberculous, since the tuberculin test can be employed to settle that when the calf is born.

THE LONDON ROAD CAR COMPANY AND GLANDERS.

ON several occasions within the last few years the columns of this Journal have been utilised to impress upon veterinary surgeons the duty of pointing out to horse-owners that the discovery of mallein has opened up a way by which glanders may, with small cost, be eradicated from any stud. The advice thus given appears to be gradually bearing fruit, and evidence of this is found in the growing demand for mallein. That agent is supplied, free of charge, from the Research Laboratory at the Royal Veterinary College to veterinary surgeons in private practice, and the demand for it has grown from

1464 doses in 1896 to 3032 doses in 1897. No doubt a considerable amount of mallein procured from other sources is also employed in this country, and there must therefore be available a large amount of information regarding the value of this new means of diagnosis. Nevertheless, very little has been published to enable one to judge of the benefits which have actually been derived from its systematic use with the object of stamping the disease out of infected studs. A recent communication on this subject from the principal veterinary surgeon to one of the largest horse-owning companies in London is therefore very welcome.

At the most recent meeting of the Central Veterinary Society, Mr W. E. Taylor read a paper on "Mallein and its Uses," in which he made some interesting statements regarding the experience of the London Road Car Company in connection with glanders. Five years ago, like every other large stud in London, this Company had glanders among its horses. During the last twelve months only two cases have occurred in the stock, and Mr Taylor considers that the disease has now been stamped out. How that most desirable result has been brought about is told in the following sentences, which we quote from Mr Taylor's paper:[1]—" The chief agent next to human intelligence in our success has been the systematic use of mallein. We protect our stud against a re-introduction of the disease by testing every new purchase with mallein, and we cleanse our stables by detecting every case by the reaction of the same agent. Not only do we do this, but every horse sent to our farm has this test applied again on his return."

It is true that Mr Taylor does not give the statistics that would have greatly enhanced the interest of this statement, nor does he expressly say that every horse in the possession of the London Road Car Company has been tested with mallein, but that may be inferred from his claim that the stud is now free from the disease, and from the series of recommendations with which he concluded his paper. These were, (1) the compulsory slaughter of glandered horses, (2) payment of fair compensation, (3) recognition of reaction to mallein as evidence of glanders infection, and (4) power to test with mallein all infected studs, and to separate those horses that react.

In the discussion that followed, the experience of the London Road Car Company was hailed as a well-timed object-lesson for the owners of other glandered studs (and parenthetically it may be observed that all the large studs in London are more or less infected with the disease), inasmuch as it pointed the way to a certain, speedy, and comparatively inexpensive method of getting rid of the disease. We have not been told what was the annual loss of the London Road Car Company from glanders prior to the employment of mallein, but it must have been something considerable, and at any rate it was heavy

[1] The Veterinary Record, 19th March 1898.

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enough to induce the directors of the Company to make the initial sacrifices in order to gain permanent freedom from the disease. They did this without the bait of compensation, and presumably they are well satisfied with the result.

But all the subsequent speakers did not read the experience of the London Road Car Company as a hint to others to go and do likewise. Mr Hunting, indeed, waxed angry with those who sought to draw this lesson from the occasion ; he thought that the present inadequate compensation would continue to be a barrier to the use of mallein, and considered that the first move must be made by the authorities, both central and local. The same speaker made pathetic reference to the imaginary case of an impecunious widow who could not afford to stamp out the disease, and would therefore bring the whole stock to the hammer. We have no doubt that glanders has been spread in that way, but, as we have on former occasions pointed out, that would soon come to an end if, after the plan adopted by the Road Car Company and a good many other studs in London, every newly purchased horse were tested with mallein.

The case of the impecunious widow has, however, very little to do with the question of compensation for glanders. The largest number of glandered horses in London are found in the stables of the wealthy Railway and other Companies, and if there had been any lingering sympathy for the shareholders of these companies, in the reduced dividends which they receive in consequence of the losses entailed among their horses by glanders, it would have been destroyed by the series of opinions which their representatives have expressed regarding the use of mallein, in response to an inquiry made on this point at the instigation of one of the London vestries. With a few exceptions these Companies will have none of mallein, or at the most they will only use it to verify the diagnosis in a case of clinical glanders. But Mr Hunting, who read their replies to the Central Veterinary Society with an accompaniment of withering comments, appeared to forget that it was for the benefit of these people that he wished the money of the London ratepayers to be spent. Besides, he ought not to have been angry with those who share with him the view that the London County Council is guilty of incredible meanness when it declines to give more than £2 for a horse that is showing decided symptoms of glanders. These Companies are merely waiting for the time when the ratepayers' money will be advanced to enable them to do, for their own benefit, what it has paid the London Road Car Company to do without assistance from the Local Authority.

Mr Hunting's opinions with regard to the equity of compensation, or the advisability of it as an inducement to notification of the existence of glanders, would appear to have undergone a process of mellowing since he published his little work on glanders,[1] from which

1 "Hunting on Glanders."

we cull the following sentences. " I cannot approve of charging the ratepayer to stimulate men in protecting themselves. . . . The horse-owner who will not give notice of the disease can be dealt with otherwise than by bribes from the ratepayer. The penal clauses of the Act are sufficient if enforced." " Glanders is incurable, its existence is a source of danger to man and other animals, therefore there is no hardship when the State compels the slaughter of diseased horses." " No horse-owner of large experience who has suffered much from glanders will contradict me when I say that the man is ' in pocket ' who destroys every case as soon as he sees it." These sentences show plainly enough what Mr Hunting formerly thought about the compensation question, and he ought not to be impatient with those who have adopted what were once his own views of the matter, and do not yet see their way to recant.

The discussion at the Central Society raised the question of the curability of glanders, and brought to light some curious opinions on that point. Mr Hunting dissented from the view that before the days of mallein glanders was generally reckoned an invariably fatal disease, and he avers that long before the days of mallein cases were cured by " sulphate of iron, beans, and copper." That may be so, but we know that before the days of mallein Mr Hunting himself did not think so. " Personally, I deny the curability of either glanders or farcy," [1] expressed his opinion on that subject in 1887. At that date also the therapeutic value of " sulphate of iron, beans, and copper, had apparently not impressed itself upon him, for he then wrote : " There are a few cases in which the symptoms disappear for a short time under medicinal treatment, but always with the same result—to become again developed, and ultimately to cause death." [2] It is not impossible that the unfortunate speaker who was taken to task by Mr Hunting for saying that glanders used to be thought an invariably fatal disease had these very passages in his mind.

The author of the paper at the Central Society, supported by Mr Hunting, maintained that M. Nocard, in the interesting article of which a translation appeared in last number of this Journal, had taught that mallein exerts a curative action on glanders. That is an error into which one would have thought it was impossible to fall, since M. Nocard nowhere in the article in question hints at any such effect of mallein. What he contends is that in almost every outbreak of glanders a large proportion of the animals that become infected never develop any clinical symptom, and ultimately recover, whether mallein be used or not.

[1] " Hunting on Glanders," p. 45. [2] Ibid, p. 43.

Reviews.

Practical Toxicology for Physicians and Students.   By Prof. Dr Rudolf
    Kobert, Late Director of the Pharmacological Institute, Dorpat, Russia.
    Translated and edited by L. H. Friedburg, Ph.D.   New York :
    William R. Jenkins, 1897.

THE translator of Dr Kobert's work on Practical Toxicology says in the
preface that he has for several years followed it in his lectures at the
American Veterinary College.   It is not a work that we should care to
recommend to the British veterinary students, though we do not doubt that
it may be useful to students of analytical chemistry, for whom we should have
thought it was mainly intended. The book has been rendered into good English,
though the spelling of a word here and there suggests that a process of differ-
entiation of the language is going on on opposite sides of the Atlantic.

The Clinical Diagnosis of Lameness in the Horse.   By W. E. A. Wyman,
    V.S., Professor of Veterinary Science at Clemson A. and M. College, and
    Veterinarian to South Carolina.   New York : William R. Jenkins, 1898.

THE author of this work has endeavoured to fill an undoubted gap in English
veterinary literature, and in his preface he disarms a good deal of criticism by
admitting that it is primarily a compilation, and that for a great deal of the
matter in it he is indebted to the German works of Professor Möller.   But a
book may be very useful although it contains little matter that is quite original,
and we can cordially recommend this one to the English-speaking veterinary
student.
    The opening chapter gives some useful directions as to the method of detect-
ing the lame leg, and the second discusses in a general way the detection of
the precise seat of lameness.   The remainder of the work is devoted to a
detailed consideration of the lamenesses of each region of the fore and hind
limbs, and those rarer forms due to morbid conditions in other parts of the
body.   The text is illustrated by a number of Figures, and we are sorry to
have to say of some of these that they do little credit to the artist.   We trust
that this defect may be remedied in a second edition.   The book is very neatly
bound and printed.

A Text-Book of Horse-Shoeing for Horse-shoers and Veterinarians.   By A.
    Lungwitz, Instructor in the Theory and Practice of Horse-shoeing, and
    Director of the Shoeing School of the Royal Veterinary College in
    Dresden.   Translated from the eighth German edition by John W.
    Adams, A.B., V.M.P., Professor of Surgery and Obstetrics, and Lecturer
    on Shoeing in the Veterinary Department, University of Pennsylvania.
    Philadelphia : J. B. Lippincott Company, 1898.

As a scientific and practical treatise on horse-shoeing, the German work of
which this is a slightly abridged translation has no equal in any language,
and it is somewhat strange that although the first edition of it appeared nearly
thirty years ago, it has only now been made available for English readers. The
translation by Professor Adams is considerably smaller than the original work,

mainly owing to the omission of the chapters dealing with the history of horse-shoeing and the minute anatomy of the hoof. Perhaps from the point of view of the veterinary surgeon, and certainly from that of the farrier, these omissions will not be thought to detract greatly from the value of the translation.

The first chapter, extending to forty pages, is devoted to a description of the horse's foot, admirably clear in respect of its wording, and made still more plain by no fewer than thirty-seven woodcuts, of which it would be impossible to speak too highly. This is followed by a chapter of twenty pages, in which the relationship of the foot to the entire limb is discussed. The remainder of the work falls into two parts, one of which deals with the shoeing of the healthy hoof, while the second treats of what may be termed pathological shoeing. The text throughout is profusely illustrated, and there is not a bad figure in the book. Professor Adams has done the work of translation well, and he is to be congratulated on having produced a work that deserves a wide sale among veterinary surgeons and shoeing-smiths in the United States.

A Handbook of Horse-shoeing, with introductory chapters on the Anatomy and Physiology of the Horse's Foot. By Jno. A. W. Dollar, M.R.C.V.S., with the collaboration of Albert Wheatley, F.R.C.V.S. Edinburgh: David Douglas, 1898.

Mr DOLLAR has already laid the veterinary profession in this country under a debt of gratitude by his translation of Möller's text-book on Operative Veterinary Surgery, and now, after a brief interval, he has, with the assistance of Mr Wheatley, made another welcome addition to English veterinary literature.

In the preparation of this work Mr Dollar has taken the German text-book by Lungwitz, above referred to, as his model, and, indeed, it may be said that the English work is mainly a free translation of the German one, with such omissions, additions, and emendations as were considered necessary, in order to meet the requirements of English readers. This is freely acknowledged in the preface, and although the plan which has been adopted is not free from objection, those for whom the book is intended need not complain, as it has produced a work that is certainly of greater value to English readers than a mere translation of the German work would have been.

The book forms a handsome volume of over 400 pages, and it is most lavishly illustrated, having an average of about one illustration for each page. These include the illustrations of the German work, supplemented by a series of admirable plates with large-sized figures of the various patterns of horse-shoes in use in this country. For these and the text descriptive of them Mr Wheatley is responsible, and Professor Mettam has contributed some pages dealing with the structure of the foot.

Putting aside the introduction and an excellent history of the art of horse-shoeing, the work falls into three sections, of which the first is devoted to the anatomy and physiology of the horse's foot, the second to the general principles of shoeing and the shoeing of the healthy foot in the various classes of horses, while the third treats of the shoeing of diseased feet and those of lame horses. The shoeing of oxen, and teaching schools for farriers, are subjects discussed in an appendix.

Space forbids that we should do more than give this bare outline of the contents of the work. We have said enough to indicate that it constitutes an exhaustive treatise on the subject with which it deals, and if it obtains the sale that it deserves it will do much to extend the practice of rational shoeing, and may, in part at least, compensate for the lack of properly established schools for the scientific teaching of farriery in this country.

CLINICAL ARTICLES.

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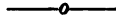
THE OPERATION OF PERONEAL TENOTOMY FOR THE RELIEF OF STRINGHALT.

By FREDERICK HOBDAY, F.R.C.V.S., Royal Veterinary College, London.

IN the *Journal of Comparative Pathology and Therapeutics* for June 1897, Mr William Robb has recorded two instances in which the operation of peroneal tenotomy was performed for the relief of stringhalt, the result in each case being very satisfactory.

Shortly after reading of these cases an opportunity occurred in the College clinique for the trial of the operation. The subject was an aged mare suffering from stringhalt in a very exaggerated form ; so much so that lately the owner had been unable to use her for cab work, and even when attempting to trot out of harness it was only with the greatest difficulty that she could make any pace. There was also a spavin on each hock.

The animal was cast, cocaine injected, and the peroneus tendon of each leg cut through. The wounds were afterwards covered with iodoform and collodion (1-12), and they healed rapidly. Upon being trotted immediately after the operation there was no perceptible improvement or alteration in gait, and the actual cautery was applied to each spavin. When returning for re-inspection a week later there were still signs of stringhalt, but the gait was perceptibly improved. The animal was then turned out to grass for about six weeks. Since that time the owner has been able to keep her in regular work, and when seen at the College a few days ago the animal could trot and turn well, only showing the stringhalt in a mild form. As in Mr Robb's two cases, there was not an absolute disappearance of the stringhalt, but the animal was transformed from a state of uselessness into a condition in which it could but put to such a severe test as the everyday work of a London cab horse.

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OUTBREAK OF DISEASE OF THE UDDER IN DAIRY COWS (CONTAGIOUS MASTITIS).

By JOHN MALCOLM, F.R.C.V.S., Birmingham.

ON returning here from London on the evening of 16th July last, I found instructions awaiting me to go as soon as possible to a farm in the suburbs and inspect and report upon a herd of cattle said to be suffering from sewage poisoning. I at once proceeded to the farm, and on inquiry was informed that a number of cattle had been poisoned by drinking water from a stream polluted with sewage through an accidental overflow from a contiguous sewer some days previously.

The herd consisted of eight cows and eleven calves, none of which

however had died, but four of the former and one of the latter were shown to me as affected.

Three of the cows were in fair condition, but were slightly fevered and suffering from acute disease of the udder ; the other was much emaciated, and had a chronic cough and old standing disease of the udder, one quarter being atrophied and another indurated.

The calf was emaciated and hide bound, and had a husky cough.

I may say here that the fourth cow and the one standing next to her, but not shown to me as one of those affected, both died, the one about a month, the other about two months, after this date.

I wished to see their *post-mortems*, and made a special request to that effect, but unfortunately no opportunity was given me of doing so. It was, however, my opinion that both were suffering from tuberculosis, and I believe both died from that disease.

The calf referred to and one of the others also died about a month after. I did not see either *post-mortem*, but it is admitted that both died from hoose.

The udder disease from which the three cows were suffering was locally manifested by a rash on the base of the teats and on the adjacent gland surface, by tumefaction and induration of the gland structure surrounding the milk sinuses, by pain on pressure on the indurated regions and during milking, and by a marked alteration in the quantity and quality of the milk, which was much reduced in amount and appeared a dirty yellow liquid containing curdled stringy clots.

In each case all the quarters of the udder were affected, but in a varying degree.

There was little or no œdema or enlargement of the udder as in cases of sporadic mastitis seen subsequent to parturition. In each case the disease was evidently in a different stage of evolution ; in one it was in the initial stage, in another it was more advanced, and in the third recovery had commenced. This was evidenced by the height of the fever, by the condition of the udder and the milk, and by the supplementary testimony of the herd in attendance as to the order of its appearance.

The appetite was considerably lessened, but there was no sign of any gastro-intestinal disturbance, and the fæces were quite natural.

I failed to trace any connection between this udder affection and sewage poisoning, for although the symptoms of the latter are perhaps imperfectly known, nevertheless there was an entire absence of those naturally to be expected in disease arising from such a cause. On the contrary, I considered the udder affection a local disease, probably contagious in character (which later on was confirmed).

As the matter was of considerable importance I desired another opinion on the subject, and at my request Mr Olver of Tamworth was subsequently called in, and examined the whole herd on 27th July. By this date one of the cows had practically recovered, and the other two were progressing favourably, but in the meantime other two cows were affected.

Mr Olver's report fully corroborated mine. The udder affection he diagnosed as a contagious local disease transmitted by the hands of the milker from cow to cow, the disease affecting the two old cows as tuberculosis, and that of two calves as hoose.

On 28th August I re-examined the cattle in company with Professor Stockman.

In the interval one old cow and the two calves had died. The five cows affected with disease of the udder had all improved in condition, and only two now showed any local indication of the disease. I narrated the whole circumstances to Professor Stockman, and from that, together with the remaining indications of the udder disease, he expressed an opinion in agreement with Mr Olver's and mine.

As soon as marked alteration in the milk was observed its sale was discontinued, and it was subsequently given to the pigs on the farm, none of which ever showed any ill effect from being fed on it.

The Medical Officer of Health was made aware of the outbreak, and naturally was on the *qui vive* for any disease that could be attributed to the milk, but no case in any person supplied with milk was brought under his cognisance.

AN INTERESTING HERMAPHRODITE.

By A. WILSON, M.R.C.V.S., Assistant, R. SCOTT & CO., M.R.C.V.S., Bombay.

THE subject of these notes was a piebald waler Galloway, six years old, imported into Bombay by an Australian dealer last cold season.

I only saw the animal on one occasion, when the owner drove into the compound, and I was so struck with the appearance of the genital organs that I made a rough sketch of the hind parts and took a few notes for my case book.

For some of the following particulars I am indebted to Vet.-Lieut.-Col. Anderson, with whom the animal was stabled, and also to Vet.-Lieut. Baldrey, who, with Vet.-Major Mills, Principal of the Parel Veterinary College, photographed the hind quarters.

The drawing shows the animal's buttocks, the anus above, three bulbous-looking bodies below, and the perinæum stretching between.

The perinæum, which resembled that of the horse, shows the outline of the urethra in the median line, till it is lost sight of close to the vulva.

The vulva was small, the labia measuring I inch or rather more from the upper to the lower commissure, and, though minus a clitoris, was otherwise perfectly formed. The vagina was immature, and the tube of the vulva ended in a *cul-de-sac*, which may or may not have been an imperforate hymen. The vulva dipped into the upper border of the glans penis, which is seen immediately below and around the vulva. The glans penis resembled that of the stallion, and protruded a little beyond the perinæal skin, the place of the prepuce being taken apparently by some loose connective tissue situated supero-laterally.

The urethra opened immediately below the lower commissure of the vulva, about the centre of the glans, and from this aperture the urine used to dribble.

On examination per rectum, moderate-sized testicles were felt in the lumbar region, and the mammary glands and teats were fully developed, as in the mare.

The penis when in a state of semi-erection projected backwards and upwards 12 inches or more.

It was very amusing to see the animal making up to a mare in season, straddling its hind legs, pressing back against the bamboos of the stall, and marking time with the hind feet.
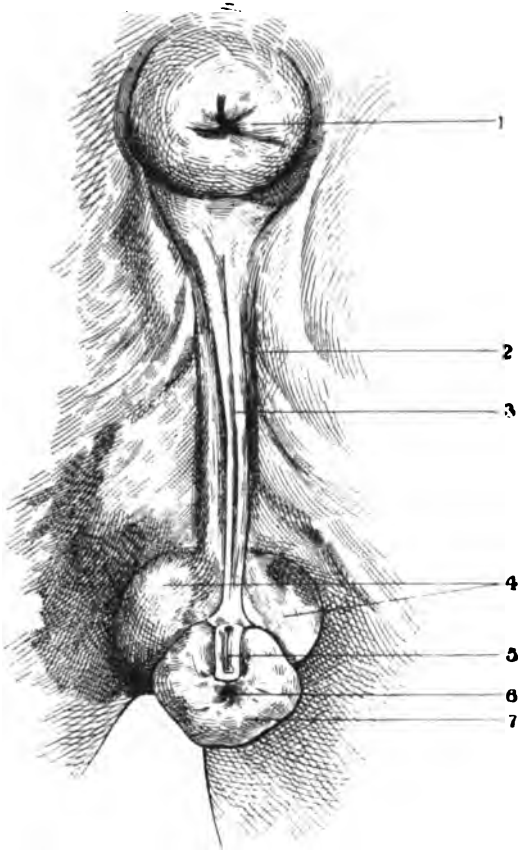
1. Anus; 2. Perineum; 3. Outline of urethra; 4. Loose tissue representing prepuce 5. Vulva; 6. Meatus urinarius, penis.

Remarks.—This case appears to me to be unique in some respects, and worthy of record.

Such strange freaks of nature are certainly of rare occurrence, and likely to be of special interest to students of development.

I might add that the animal was in good condition, and was driven daily by the importer, till it was sold in a batch up-country and lost sight of.

NOTES ON TUBERCULIN REACTION.

By HAROLD SESSIONS, F.R.C.V.S., F.H.A.S., Brighton.

FROM observations I had made on cattle on farms where I had been a pupil, and later on cattle of my own, I came to the conclusion that a large number of cases of tuberculosis could be arrested, and that recovery might take place, if the cattle were placed in a bracing atmosphere and lived an exposed and open-air life. This statement does not apply to advanced cases, as in some of these I noticed that the exposure was more than the animal could physically bear, and seemed to accelerate death. In a paper which I read on 24th January 1895, the following paragraphs occur:—

" I believe that large numbers of cattle recover after an attack of tuberculosis, and these animals will remain with the portion of their

| No. | 1897 | | | | | | 1898 | | | | |
| | February 3 | | February 4 | | | | Mar. 1 | Mar. 2 | March 3 | | |
	11 a.m.	10¹ p.m.	6 a.m.	9 a.m.	12 noon.	3 p.m.	10.30 a.m.	8² p.m.	5.30 a.m.	10 a.m.	2 p.m.
II. . .	101·8	101·0	101·3	101·5	102·2	103·6	101·4	101·2	101·0	101·2	101·8
VIII. . .	101·2	101·4	100·8	101·0	101·3	101·4	101·4	101·2	101·0	101·6	101·2
IX. . .	102·1	102·5	103·3	106·5	107·6	105·3	101·2	101·2	102·0	103·6	104·6
X. . .	102·0	102·0	102·2	104·1	105·4	105·5	101·2	101·4	101·4	101·6	102·2
XIII. . .	101·5	100·8	104·6	105·0	104·1	103·4	101·4	101·2	100·6	101·4	101·6
XVI. . .	101·8	102·0	102·2	102·8	105·2	106·0	101·4	101·0	101·2	101·6	101·8
XVII. . .	101·3	101·8	101·4	102·2	105·5	105·7	101·4	101·2	101·0	101·6	101·2
XIX. . .	101·3	101·4	100·6	100·0	101·1	101·2	101·4	101·2	101·0	101·6	101·2
XXV. . .	102·0	101·8	102·6	106·0	107·4	105·4	101·0	100·6	101·0	102·0	101·8
XXVIII. . .	101·5	100·5	101·4	102·9	106·0	105·3	102·2	101·2	101·4	101·2	101·4
XXXIII. . .	102·4	101·1	101·0	101·3	101·4	101·3	101·2	101·2	102·0	103·6	102·2
XXXIV. . .	102·0	101·8	101·6	102·6	106·1	104·4	101·2	101·2	101·0	101·2	101·4

¹ Tuberculin injected.   ² Tuberculin injected.

body attacked by the disease injured and altered to the end of their lives. But the actual disease has been overcome.

" If before the disease is far advanced the cow is placed in the open air, fed on good nourishing but not stimulating food, particularly food of an oily nature, the chances are the disease will be stopped, and will not develop further unless she is again subjected to unhealthy surroundings."

Since I wrote these paragraphs I have met several personal friends

who have undergone a similar treatment for tuberculosis, and who have now recovered. The tubercle bacilli in their cases was found before the treatment commenced.

For some years I have tested a herd of cows with tuberculin, and the table of temperatures (p. 90) is very interesting. In 1896 these cows were standing amongst animals that had reacted, but were all apparently sound. In 1897 they were kept in a low house with very little ventilation, and the inside temperature was rarely below 60° F. to 65° F. They were highly forced for milk, and did not go to pasture. In the spring of 1897 this was changed. They were given plenty of ventilation when indoors, but most of their time was spent on some hill pastures on the sea coast, where the air is particularly bracing.

The last testing was carefully carried out, and on the same night from the same lot of tuberculin I tested a number of other cows and had reactions up to 106° F.

I wish to place these facts on record without attempting to explain them, hoping that in the course of time other temperature charts extending over a series of years may be available for comparison. In the Castlecraig herd I believe the cattle have had almost identical reactions year after year, and have not reacted in the manner which those I record have done. Of the twelve I have followed up, six have not reacted a second time, three react slightly or hardly at all, two have never reacted, and one reacts in 1898 which did not in 1896 and 1897.

A CASE OF ŒSOPHAGEAL ULCERATION IN THE HORSE.

By F. B. JONES, M.R.C.V.S., Leicester.

THE following case may be of interest as an example of œsophageal obstruction, apparently originating in ulceration of the mucous membrane of the gullet.

I was first called to see the animal, an aged cart gelding, on the 9th of March, and while I was examining him he made several spasmodic efforts to vomit. During these attacks he generally knelt down, and vomited about a pint of thick mucus and saliva. The attacks occurred at intervals of an hour or less, and between them the animal appeared to be free from pain. On inquiry I learned that the horse had shown symptoms of choking about three months previous to the present attack. He had, however, been working up to the 8th March, on which day the carter thought he was not quite right as he slobbered at the mouth a good deal.

My diagnosis was obstruction of the thoracic portion of the œsophagus, and I ordered a pint of water to be given every two hours. This, however, brought on attacks of pain and spasms, and I therefore ordered it to be discontinued.

As the horse was of little value, the owner had him killed on the 11th March, without my knowledge. Fortunately, however, I obtained the gullet at the knacker's next day, and on slitting it open I found

THE RINDERPEST IN SOUTH AFRICA.

By F. A. VERNEY, M.R.C.V.S., Natal.

ON my arrival in Natal at the end of November 1896 I was at once instructed to proceed to the Transvaal to assist Messrs Watkins-Pitchford and Theiler, principal veterinary surgeons of Natal and the Transvaal respectively, in their experiments with rinderpest. After twelve days' travelling I arrived at the experimental camp, which had already been organised about six weeks. This camp was situated near the Marico River, about 20 miles from the British Bechuanaland border, and 150 miles north-west of Pretoria. The latter distance had to be traversed by a bullock waggon over what in England would be considered an impassable road. For our letters and provisions we had to send 96 miles, which often caused us to be without the necessaries of life for about twenty-four hours. Under these conditions it is needless to say experiments had to be carried out under the most adverse circumstances—circumstances sufficient to daunt the most energetic.

In attempting to find something valuable as a prophylactic or curative agent for rinderpest, Messrs Pitchford and Theiler paid particular attention in the direction of serum-therapy, a treatment destined to save thousands of cattle in South Africa. As the experiments proceeded, it was discovered that large doses of serum obtained from an animal that had recovered from rinderpest only conferred a passive immunity, at the most for a month or so. When this fact was definitely ascertained, the object of Messrs Pitchford and Theiler was to convert this passive immunity into an active one, an object they accomplished by giving the serum and the virus of rinderpest simultaneously, the serum counteracting the virus to such a degree as to give the animal a modified form of the disease, by

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which an active immunity was conferred. As time has progressed, and thousands of cattle have been treated, considerable improvements in the serum-therapy of rinderpest have been made, and even to-day there are vast improvements wanted.

In assisting at the experimental camp I had excellent opportunities of observing the rinderpest of South Africa in all its stages. Comparing the disease with that described in the English text-books, the symptoms are somewhat similar, that is to say, one could diagnose a case of rinderpest fairly well from following English text-books. With the *post-mortem* appearance it is quite a different matter. Being well acquainted with the English accounts of the lesions of rinderpest, I was greatly surprised when I made my first *post-mortem* examination on an animal that had died of undoubted rinderpest. The following is a description of the lesions found :—

Yearling bull ; carcase much emaciated but skin normal. Mouth, tongue, and œsophagus normal. Rumen showed a considerable number of the amphistoma conicum ; otherwise normal. Reticulum and omasum normal. Abomasum showed inflammation of the mucous membrane, with hæmorrhagic areas varying in size from a pinhead to a pea. About 6 feet of the jejunum showed an intense enteritis, and the mucous membrane throughout the small intestines showed hæmorrhages up to the size of a hazel nut. The mucous membrane at the apex of the cæcum inflamed. Large colon showed four well-marked ulcers and enteritis. Rectum showed inflammation of the mucous membrane, with what are called "zebra markings." Intestinal lymphatic glands enlarged and congested, some on section showing a light chocolate-looking liquid. Spleen normal. Kidneys normal. The mucous membrane of the bladder showed a few small ecchymoses. Liver showed evidence of biliary congestion ; gallbladder full ; bile normal in appearance. Lungs showed areas of emphysema and collapse. Heart normal. Septum nasi and turbinated bones markedly inflamed. Brain normal.

What struck me as the most remarkable dissimilarity to the English rinderpest was the fact of the rumen, reticulum, and omasum being normal, and in no case have I ever found any well-marked change in these stomachs.

In making a large number of *post-mortem* examinations one is struck with the variations one finds in each animal. One may make ten *post-mortem* examinations and not find two alike. Sometimes one may find the abomasum intensely inflamed and ulcerations well marked, with only slight alterations in the intestines, and, on the other nand, one may find the abomasum inflamed, though not intensely, while the whole of the intestines are acutely inflamed and ulcerated, with in some cases portions of the mucous membrane in a diphtheritic condition, similar in appearance to that seen in swine fever. Sometimes the inflammation of the intestines is chiefly located in the small intestines, sometimes in the large.

After finding these numerous differences, I endeavoured to ascertain, by making a large number of *post-mortem* examinations, what were really the most constant lesions of the South African rinderpest, so that one might when making a *post-mortem* examination positively say whether it was rinderpest or not. After a great deal of work I came to the conclusion that the constant and positive

lesions of the rinderpest of this country consisted in inflammation of the nasal cavity and abomasum, either ecchymoses or inflammation of the mucous membrane at the blind end of the cæcum, and "zebra markings" of the rectum.

At the end of March 1897, owing to the behaviour of the Transvaal Government, our camp was broken up, and Mr Pitchford and myself came back into Natal, where rinderpest, although it was prevalent in the Orange Free State and Transvaal, had not gained a foothold. It was not until the middle of July that the first outbreak occurred in this colony.

For two years our borders had been protected by means of a double fence, regularly guarded, a procedure which cost the Natal Government nearly £100,000; and there is good reason to believe that had not rinderpest been sown broadcast throughout the colony by our malicious neighbours we might have been free of the disease to-day.

When the first outbreak occurred, our first endeavour was to stamp out, but owing to so many outbreaks occurring directly after, and in so many different parts of the colony, such procedure was soon found to be entirely impracticable.

Not being able to continue the stamping-out policy, our next aim was to treat the disease. The methods in use were Dr Koch's or the bile method, and the serum or Pitchford-Theiler method. The wish of the Veterinary Department was to use the serum method, but at first this was quite impossible, as there were no salted[1] cattle in Natal to obtain serum from. The bile method had consequently to be used. In carrying out this treatment Dr Koch's instructions were stringently adhered to, inasmuch as my colleagues, Assistant-Veterinary Surgeons Stapley and Baxter, had visited Dr Koch at his experimental camp at Kimberley, and were thus acquainted with every detail of the work.

Dr Koch's method consisted in taking the bile from an animal that had been suffering from rinderpest four or five days, or even from an animal that was on the point of death from the disease. The bile was to be taken with every antiseptic precaution, taking care when puncturing the bladder with the trocar and canula not to wound a blood-vessel. The bile to be selected for use was to be of a green colour, clear looking, frothy, having a sweet smell, and containing no sediment. For a full-grown animal 10 cubic centimetres were used. It was introduced subcutaneously at the dewlap, and one inoculation was deemed sufficient. Dr Koch claimed that such an inoculation would confer an "active" immunity, and he further stated that the bile from a rinderpest animal could not confer the disease. He also stated that bile had no curative properties, and was of little use once rinderpest had gained access to the herd.

Bearing all these assertions in mind, the Veterinary Department went to work with all possible speed, and advised farmers accordingly. But as time went on the work did not progress so satisfactorily as one could have desired, and experience has shown us that the bile method as taught by Dr Koch is a failure.

In the first place, it is impossible to confer an active immunity with one inoculation of bile, and it seldom confers immunity for

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[1] The term "salted" is applied to cattle that have suffered from rinderpest and recovered.

more than four months, sometimes not for longer than twenty days.

The statement that bile cannot confer disease is entirely erroneous, as many herds after inoculation break out with rinderpest to a greater or lesser extent, notwithstanding that they are perfectly free from the disease at the time of inoculation. Whether bile can confer disease or not proved a most important question, for being under the impression that it could not, one was justified in advising farmers to inoculate their cattle in absolutely clean areas. Inoculation with bile has been the means of introducing rinderpest into parts where it might not have been established even to-day, and it has thus caused the loss of thousands of cattle.

Notwithstanding the want of success obtained by using bile according to Dr Koch's instructions, bile undoubtedly can be used with fairly successful results. To obtain these one should use no other bile than that obtained from an animal that has recently died of rinderpest, and in taking it one can use a bile that is yellow or brown as well as the green, and it need not necessarily be clear-looking— in fact, all biles may be used except those having an offensive odour and a large amount of sediment.

There is no doubt that all biles are not of the same strength, which teaches one the importance of mixing all the biles obtained together. The dose to be used is from 5 cc. to 10 cc., according to the size of the animal, and in all cases one should carry out the inoculations at an interval of ten days. The reason of this is because of the uncertainty of the action of bile, as there is no doubt that some samples have not much more effect than ordinary water. Hence, if one is unfortunate enough to obtain such a bile, by using two inoculations this chance is minimised very considerably. In suggesting this method of procedure as successful, I do not mean to assert that it is infallible, but it is the most likely to produce an immunity of several months.

With regard to the curative properties of bile, there is no doubt that it sometimes has curative properties. This I have often seen proved. If 50-100 cc. of bile taken from a recently dead rinderpest animal be introduced intravenously into a sick animal recovery frequently takes place. Such a procedure sounds highly dangerous, and one cannot help being struck with the mixture of blood and bile, but I have not seen anything serious follow. The curative property of bile justifies one in using it in a herd in which rinderpest has already broken out, and I have frequently seen the disease suddenly stop after introducing the bile.

This curative action of bile is opposed to the belief that the bile confers immunity because of its being an attenuated virus, for undoubtedly in many cases it possesses antitoxic qualities. In considering the bile method one cannot help being surprised at the great errors which Dr Koch committed regarding its effects and actions. For one month the Veterinary Department of Natal was carrying out the bile inoculation, and during this time cattle that had recovered from rinderpest were procured from the adjoining States, which enabled us to stop bile inoculation, and adopt serum inoculation—a method we knew would certainly confer an active immunity.

In procuring salted cattle it was all-important that animals that

had suffered from rinderpest in a severe form should be obtained, as it is only natural to suppose that these animals yield a better serum than those that have had only a mild form of the disease. In obtaining such cattle one had to depend upon the honesty of the farmers in the adjoining colonies, but unfortunately in many cases it was evident that such a quality as honesty was not recognised. There is no doubt that many of the so-called salted cattle were simply immunised, and, in course of time, they developed rinderpest. The serum obtained from such cattle naturally was responsible for many deaths in the serum treatment. Another difficulty also arose from the fact that Transvaal and Free State cattle are most susceptible to redwater when brought into Natal, and as the parasite of redwater resides to a great extent in the blood, there was great danger of conferring this disease when treating animals for rinderpest. Having obtained suitable cattle, the first thing is to prepare them for bleeding.

If the animal has only recovered from rinderpest four or five weeks, then for the first bleeding it is not necessary to introduce virulent blood, but, on the other hand, if the animal has recovered for a longer period than this, it is advisable to give it an injection of from 50-100 cc. virulent blood, and wait five to seven days. When this period has elapsed, and the temperature is normal, one can consider the animal suitable for bleeding. In obtaining virulent blood, there are certain precautions one should carry out. There should be no doubt that the animal is suffering from rinderpest, and this disease only. It is always advisable to obtain blood for the inoculation of salted cattle from animals in the early stages of the disease, and having a temperature of 104° F. or over. The reason why blood should be taken from animals in this condition is that, in the later stages of the disease the blood, though taken with strict antiseptic precautions, frequently gives rise to serious swellings at the seat of inoculation, which, I think, is due to the presence of putrefactive and other organisms that appear to be present in the blood of a rinderpest animal in the late stages of the disease.

Some workers, instead of introducing the virulent blood subcutaneously, introduce it intravenously ; but this I object to, as one cannot always personally obtain virulent blood, and therefore cannot depend upon its being aseptic. I think it is better to have a local abscess than to set up a pyæmia.

Some workers give very large doses of virus (up to 1000-2000 cc.), but I do not favour this method, on account of its impracticability when one has a large herd of salted cattle to be systematically inoculated. Moreover, I have the best of reasons for believing that one can obtain results equally as good when 100 cc. are introduced systematically. In treating the salted cattle under my own supervision, which numbered about one hundred, I have always endeavoured to give them 100 cc. subcutaneously every six or seven days, and I think I may safely say the results obtained from the serum supplied by cattle treated thus compare favourably with any in the colony, notwithstanding that some of our department have carried out the method of large doses of virus given intravenously.

Having brought the animal to a condition suitable for supplying reliable serum, one may then bleed it. The method employed is as follows :—The animal is secured by casting, and fastening all four legs together, and the legs are then pulled outwards and backwards, the head being tied to a ring in the ground. A small cord is now passed around the base of the neck and tied sufficiently tight, so as to raise the jugular veins. A portion of the skin over the jugular vein is then washed with soap and a solution of creolin, and shaved with a razor, after which a solution of perchloride of mercury is poured over the shaved area. With a sharp scalpel a small incision is made through the skin along the line of the vein, and a trocar and canula, having a diameter equal to an ordinary-sized lead pencil, are introduced into the vein, the direction being either towards the head or the heart, whichever one prefers. The blood is received into glass jars, the capacity of which is marked at intervals. The amount taken varies according to the size and condition of the animal, but from one of average size 4000 ccm. can be safely taken. When the desired quantity is abstracted, the cord around the neck is untied, the canula at the same time being withdrawn. The neck is then washed with a creolin solution, followed by a solution of perchloride of mercury, and the animal is released.

If the animal is in good condition, it may be bled every seven days, providing that after each bleeding it receives virulent rinderpest blood. The treatment of the blood when withdrawn depends upon whether one is going to use defibrinated blood or serum. One is equally as good as the other, but for immediate use the advantage lies with the former, as it is much more quickly and economically prepared. In preparing the defibrinated blood, it is emptied into enamelled buckets which have been previously boiled, and it is then sharply whisked with an iron brush previously sterilised by heat. After ten to fifteen minutes whisking the blood is strained through sterilised muslin into bottles, and then it is fit for use. The dose of defibrinated blood varies according to when and at what stage of the disease it is employed, but, roughly speaking, it varies from 50 ccm. to 500 ccm.

In using defibrinated blood it is of the utmost importance that it should be used soon after it is withdrawn, always within eight hours. If kept longer than this period it is very liable to cause serious swellings at the seat of inoculation, notwithstanding that it has been taken with every antiseptic precaution. The swelling is apparently due to a particular organism, as the character of the swelling is nearly always the same, namely, emphysematous, and accompanied by a gelatinous degeneration of the subcutaneous and adjoining tissues. If this swelling is severe it often causes death, unless heroic surgery is carried out, and the diseased parts well exposed.

In preparing the serum the blood is placed in enamel buckets and allowed to stand for twenty-four hours, the serum being drawn off by means of a pipette or a syringe. The dose of serum compared with defibrinated blood is usually reckoned as half. The advantage of the serum is that it will keep longer than defibrinated blood, and is not so liable to cause swellings. The fact of defibrinated blood not retain-

ing its aseptic properties for any length of time led Mr Watkins-Pitchford to endeavour to discover a preservative, which he was successful in doing in the shape of formalin, added in the proportion of one-third per cent. By adding this, defibrinated blood can be kept for almost an indefinite time, but in some cases the blood forms into a soft crassamentum.

This preservative also answers well for serum, or one may use a half per cent. of phenol. Having obtained the defibrinated blood or serum, then the next thing is to use it. The dose employed varies according to the circumstances, that is, whether one is about to treat a clean herd of cattle, or cattle that are suffering from the disease. In speaking of the doses of serum I refer to defibrinated blood, which has been almost exclusively used by the Veterinary Department. In treating a herd not affected with the disease the following procedure, after a large experience by the Natal Veterinary Department, has been found the most satisfactory.

Twenty-four hours before giving serum the herd is inoculated with 1-2 ccm. of virulent rinderpest blood, and when this time has elapsed serum is given subcutaneously in doses varying from 25 ccm. to 100 ccm., according to the size of the animal. On the fifth or sixth day the temperature of each animal is taken, and if over 104° F. a second inoculation of serum is used, varying from 75 ccm. to 300 ccm. In nearly all cases the rise of temperature takes place on the fifth day, and the external symptoms commence to manifest themselves on the sixth day, and it is at these periods that a large dose of serum is required. Many give the second inoculation on the fifth day, but personally, I prefer the sixth day, as often by giving the serum at the former time one cuts the disease completely short, and it is questionable if an animal that has only had a thermometric reaction to the virus of rinderpest possesses an active immunity, whereas if one gives the second inoculation on the sixth day in most cases there is not only a thermometric reaction but external symptoms. When an animal recovers after having shown these, one is justified in calling such an animal "salted." On the other hand, by giving the serum on the sixth day, one does not save so many animals, but I think a herd treated in this way with a recovery of 75-80 per cent. is better than one with a recovery of 95 per cent., but with many of the animals only possessing a passive immunity.

Herds treated in this systematic way vary as to the intensity of the disease which they contract, which is perhaps due to the degree of natural resistance which they possess, or more probably due to the quality of the serum employed. When the animals so treated show the disease somewhat severe, then it is advisable to give a third inoculation, or even a fourth, using larger doses of serum, varying from 200 to 500 cm.

When a herd of cattle is treated as described, 75-95 per cent. usually recover, but the weather and surroundings have to be taken into consideration. Wet weather is distinctly prejudicial to the recovery of a rinderpest animal; also if an animal is allowed to have a liberal supply of water. Keeping a number of sick animals in close quarters is undoubtedly adverse to recovery.

When serum was first employed in treating a healthy herd of cattle it was customary to introduce the virulent blood and serum at the

same time, with the result that many of the animals did not become sick, but were simply immunised. This fact put cattle-owners to a lot of trouble extending over a long period of time, and consequently the Veterinary Department adopted the method of introducing the virus first.

In the case of animals that have already contracted the disease, one has to carry out energetic treatment and use large doses of serum if success is to be obtained. The chance of success depends on the stage of the disease at which the sick animal is treated. If the animal has only shown a rise of temperature, and external symptoms are just beginning to manifest themselves, then one has in many cases good hope of success, providing reliable serum is obtainable; but if the disease has existed for two or three days or more, then the animal usually dies. An animal that has had no previous treatment with serum, but has been inoculated with bile at some previous time, often has a less severe form of the disease than one that has had no previous treatment with bile.

In treating a sick animal doses varying from 100 cm. to 500 cm. are used, and repeated daily until the animal shows evidence of recovery. I always adopt the plan of giving half intravenously and half subcutaneously, as I am of the opinion that an intravenous injection is quicker in its action than a subcutaneous one—a matter of considerable importance in a case of rinderpest.

Although the Veterinary Department have almost exclusively used defibrinated blood, there are occasions when serum only must be used. This is when imported cattle are treated, as nearly all colonial cattle possess the casual agent of redwater in their blood, and as this agent resides chiefly in the red corpuscles one considerably minimises any chance of giving the animal under treatment redwater by using serum.

With regard to the serum itself there are many interesting points.

It is an undoubted fact that two animals, notwithstanding that they have both had the disease in a most severe form, will not yield serum of the same strength, and there is no doubt that the serum of some animals that have had an acute and prolonged attack of the disease has very little immunising or curative properties. These facts teach one the importance of always mixing the serum of each animal bled—a procedure I always endeavour to carry out.

Another fact regarding the quality of the serum is that as time elapses after recovery from the disease so does the quality of the serum deteriorate, particularly if the animal does not receive systematic doses of virulent rinderpest blood, and in time the serum of most animals apparently has no more effect in combating the disease than so much water. This is a curious fact, as one would expect that when an animal's blood became in such a condition he would no longer be able to resist the disease, but such is undoubtedly not the case.

The time when an animal yields the best serum is from one to three months after recovering from the disease, and after this time the serum in most cases gradually, and in some cases rapidly, deteriorates in quality. I have some animals in my possession now that suffered from rinderpest seven months ago, and their serum to-day has little

or no beneficial effect, notwithstanding that these cattle at a previous period have furnished excellent serum.

Animals after apparently making a good recovery from rinderpest often suffer from a relapse, which usually terminates fatally, serum apparently having little effect in these cases. These relapses may take place from fourteen days to three months after evidence of recovery.

Lately I have had an opportunity of making a number of *post-mortem* examinations on cattle that had recovered from rinderpest, apparently completely, but careful examination of the mucous membrane of the abomasum and intestines frequently reveal ulcers undergoing the process of healing, reminding one of what is often seen in the ulcerated intestines of a pig suffering from swine fever.

An important question to the South African farmer is, how long are the so-called salted cattle immune against rinderpest?

Recently there have been very grave rumours that salted cattle had taken rinderpest a second time, consequently I was instructed by the principal veterinary surgeon to proceed to the Transvaal, where the rumours were most current, and investigate. Notwithstanding that rinderpest had existed for eighteen to twenty-four months in this state no authentic case could be discovered.

Taking the information obtained from Russia on the subject, there is reason to believe that a salted animal has an immunity for five years, which is practically speaking for life.

In conclusion one cannot help being struck with the fact that during the last eighteen months in which rinderpest has prevailed in South Africa more has been found out regarding its treatment than during the thousands of years for which there is good reason to believe rinderpest has existed. The results achieved are due to the valuable experiments carried out by Dr Koch, and Messrs Watkins-Pitchford and Theiler.

ON THE PATHOLOGY OF SOME SPECIFIC GRANULO-MATA IN HORSES AND CATTLE.

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To define the exact position of specific granulomata in a pathological classification is a matter of difficulty, because of the resemblance which a new formation of this sort has to some of the results of what is known as chronic inflammation on the one hand and to that which we understand as definite tumour formation on the other. And at the commencement it may be said that in what follows the term "specific granuloma" is to be taken as meaning the same thing as the more commonly used "infective granuloma," the reason for preferring the use of the former being alluded to in due course.

Histologically a granuloma appears as a circumscribed collection of lymphoid cells or leucocytes of an indifferent character which are imbedded in a fine stroma or ground-substance formed either by a fibrinoid degeneration of the original tissue in which the collection of

cells has taken place, or by an exudative process from the cells them-selves, or by a combination of both processes. In a granuloma together with definite well-formed lymphoid cells will be found other, and originally similar, cells in various stages of degeneracy, and nuclear débris remaining from yet other cells which have undergone disintegration. And further, it will be noticed that these changes are more particularly in evidence at the central portion of the granuloma, in the part which is most remote from vascular sources of nutrition, than in the peripheral portion where the cells are more natural in appearance and in nearer relation to a blood supply. Besides these indifferent cells two other elements will frequently be found in the new formation, the so-called "giant cells" and an infective parasite, the presence of which has caused the granuloma. As to the import of giant cells widely divergent views are held by pathologists. Some, following Baumgarten, look upon these elements as constituting a stage in cell degeneracy, and regard their presence as significant of failure of the resistant powers of the tissues against an infecting agent. But others, with Metchnikoff, believe that a giant cell is an actively resistant element of full vitality, that it is the expression of a successfully defensive reaction against an invading parasite. The arguments in favour of the latter opinion, which the writer him-self holds, may be arranged along three lines; (1) the resemblance between these giant cells and similar cells found either in normal developing tissue with circumstances which indicate that they are the seat of physiological activity, such as the myeloplaxes of Robin, or again with like cells which are found in conditions of disease of either natural or artificial production in which it would seem probable that they have come into existence for protective purposes; (2) the fact, which seems fairly substantiated, that an active proliferation by division of such cells occurs; and (3) the fact that such cells may be found in large numbers in the lesions of healing tuberculosis.

A granuloma thus formed is usually after a short time marked off from its surroundings by a definite capsule of fine fibroid tissue. The normal tissue in its immediate neighbourhood is usually of natural appearance, except sometimes for a greater or less degree of round-cell infiltration, which, however, in the absence of secondary inflammatory processes, is not usually a prominent feature.

Individual granulomata do not as a general rule tend to increase beyond a certain comparatively small size, but larger masses may occur from the coalescence of neighbouring formations. The blood supply of a granuloma is always scanty; in the earlier stages small vessels may be seen amongst the larger cells which constitute the peripheral portion of the new formation, but the central portion is always evascular. A result of this deficient blood supply is the caseous form of degeneration which is frequently seen.

A specific granuloma may be described as one of the manifestations following the infection of an animal by a parasite whose immediate and local action on the tissues is not characterised by a high degree of virulence. And to illustrate my meaning it will be useful to briefly, and very generally, compare the formation of a granuloma in the course of infection by, for example, Koch's tubercle bacillus with that of an abscess caused by a parasite such as staphylococcus pyogenes aureus. In either case the parasite comes to rest at some

point in the tissues, and in either case the invaded area becomes at once densely crowded by leucocytes. In the case of the pyogenic coccus the bio-chemical action of the parasite upon the collected cells is such that a rapid necrosis resulting in suppuration occurs. With the tubercle bacillus, on the other hand, the local action of the parasite is much less severe. No such acute cell-necrosis occurs, but when the collected cells do die it is apparently as the result of an accidental deficiency of nutrition, rather than of any direct action of the parasites which are amongst them.

These granulomata, then, are manifestations of an infective disease caused by a specific parasite, and it is preferable to call them "specific" granulomata rather than "infective," since the tissue proper of the new formation is not infective although the parasite which causes it is. This distinction, which may at first sight appear a little pedantic, is, I think, really necessary because of certain views which some pathologists at present hold. The views which are referred to suggest for malignant disease, carcinoma and sarcoma, an origin in which either the lowest forms of animal or, according to more recent theories, of vegetable parasites have a causative influence. Thus a carcinoma may truly be described as an infective tumour, since, whatever be the cause of the primary growth, there cannot be any doubt that secondary growths are the result of direct infection by the actual tissue cells of the original formation. In a case of columnar-celled carcinoma of the larger bowel in man, for instance, we may find secondary growths in the liver which are composed of columnar epithelial cells. And in such a case the one thing of which we may feel reasonably certain is that the essential cells which form the latter growths are derived directly from the cells of the primary intestinal growth. For it is inconceivable that the presence of a parasite alone could cause the development in the liver of specialised epithelial cells such as those whose overgrowth caused the primary tumour. Multiple specific granulomata on the contrary are undoubtedly due to the dissemination in the tissues of parasites which have the property, wherever they may come to rest in the tissues, of causing new formations composed of indifferent cells. The sarcomata again, composed as they are of what may fairly be described as indifferent or non-specialised cells, would seem to be of parasitic origin far more probably than the carcinomata.

Diseases in the course of which granulomata may develop are caused by members of each of the three principal subdivisions of the vegetable parasites which are known to infect man and lower animals. Thus granulomata may be due to an infection by schizomycetes or fission-fungi, by hyphomycetes or mould-fungi, and by blastomycetes or yeast-fungi. Actinomycosis, further, a disease characterised by the formation of granulomata, is caused by the unclassified actinomyces or ray-fungus. Again, in syphilis of man we have an infective disease in which granulomata are of frequent occurrence, but of which the causative agent is still unknown.

The formation of granulomata in the course of infection by various schizomycetes is a pathological occurrence which is sufficiently well known, and it will not be necessary to more than just refer to tuberculosis and glanders as common types of such diseases.

Botryomycosis caused by a parasite of this class may, however, be

specially mentioned in dealing with granulomata occurring in horses. This disease was first accurately described by Böllinger, and later by Rivolta. The parasite causing it was studied by Rivolta and by Rabe, and has been known variously as *Botryomyces, Micrococcus ascoformans,* and more commonly as *Micrococcus botryogenus.* The parasites occur in the granulomata which are characteristic of the disease in separate discrete colonies, each of which is encapsuled in a hyaline membrane. These colonies collected in masses form largish mulberry-like bodies of a yellowish colour, and as such can be readily seen with the naked eye in the new formations. The disease has been especially recorded as an infection following the castration of horses, the granulomata developing along the spermatic cord and sometimes causing tumours of considerable size. Independently of castration the disease has also been observed as causing multiple granulomata in the subcutaneous connective tissue. During the progress of the disease similar new formations may appear in the lungs and elsewhere. Rieck has recorded an interesting case of widespread botryomycotic infection in a mare. In this case the granulomata were found on the diaphragm, and in the lungs, spleen, liver, and mesenteric glands. The presence of the disease was particularly noticeable in the uterus and ovaries, and was believed by Rieck to have been directly communicated to the mare by a stallion which was suffering from botryomycotic disease of the penis. Botryomycotic infection has also been observed in the mammary gland of the cow, and in the pig after castration.

There are, however, certain cases of specific granulomata occurring in horses and cattle as the result of infection by mould or yeast fungi to which I wish more especially now to refer. Such infective diseases have only received adequate attention during the last few years, and but few well-authenticated cases have as yet been recorded. But it is very probable that these infections are not by any means so uncommon as present records would indicate, and it cannot be doubted but that when certain difficulties in the identification of parasites of this description as they occur in the tissues have been overcome by experimental research we shall find that the field of pathology has wider limits in this direction than has hitherto been suspected. As to mould infections our knowledge, except with regard to the one species, *Aspergillus fumigatus,* is at present very fragmentary. The first occasion on which the presence of a mould parasite as a possible factor in disease was mentioned appears to have been in 1815, when Mayer described such a fungus as occurring on the lining membrane of the air passages of a jackdaw. After Mayer a number of isolated observations dealing with the occurrence of mould parasites in the living body were recorded. These were chiefly cases in which such fungi were found growing on the lining membrane of the air passages in various birds, such as ducks, hawks, bullfinches, parrots, pigeons, etc. In 1856, Rivolta recorded a case in which there was a similar appearance in a horse. In 1876, Pech found a mould in the lungs of seven horses who had fed on mouldy chaff, and one or two similar cases in cows were recorded. In all of these cases, because of the position in which the fungus was found in connection with the air passages, it might obviously have been present as an accidental secondary infection of a previously

diseased mucous membrane. The moulds described as thus occurring were *Aspergillus micheli, Aspergillus glaucus, Aspergillus nigrescens, Aspergillus fumigatus, Mucor racemosus, Mucor conoideus,* and *Mucor mucedo.*

In 1884 Martin recorded a case in which there appears to have been a general infection by the fungus, that of a horse in which in addition to a mycosis of the lungs there were numerous nodules in the liver. In the two following years Roeckl and Piana recorded each a case of infection in the cow, and Rivolta a case of general infection in a bitch by a mould which he named *Mucoromyces canis familaris.* In 1890 Dieulafoy, Chantemasse, and Widal published an extensive and careful investigation of an infective disease in pigeons, and showed that granulomata which developed in the course of it were due to the presence of *Aspergillus fumigatus.* Mazzanti in 1891 described a case of pulmonary mycosis in a lamb, but was not able to identify the species of the fungus. The course of an infection by *Aspergillus fumigatus* has been made familiar to us by the valuable and interesting researches of Lucet and Rénon. These writers have clearly proved that a natural primary infection by this parasite occurs in man, in horses, in cattle, and in many species of birds. A disease of hen's eggs which appears during incubation has also been shown by Lucet to be due to the same cause. Experimental inoculation of guinea-pigs and rabbits produces a general infection, in the course of which granulomata develop. These nodules are to the naked eye indistinguishable from similar formations occurring after infection by Koch's tubercle bacillus, and it is only after microscopic examination and cultural experiments that the distinction between the two can be made. In horses and cattle the disease may assume an acute, a sub-acute, or a chronic form. In acute and sub-acute cases the infection may run its course as a hæmorrhagic septicæmia. In the chronic form there is the formation of granulomata and a course of disease which closely simulates tubercular infection.

In the cow in particular a pulmonary aspergillar mycosis may run a clinical course which would seem to be indistinguishable from a pulmonary tuberculosis. And equally in man the difficulty in the diagnosis between the two conditions exists so far as clinical symptoms are concerned. This aspergillar mycosis is the only example of an infection by a mould-fungus the exact pathology of which has been satisfactorily worked out. Rénon and Drouin have recorded a case of what they describe as "une mycose sous-cutanée" in a horse. The animal developed a number of subcutaneous tumours, some of which had in the course of two months reached the size of a human head. The skin became adherent to them, and subsequently many of them broke down. When a tumour was completely extirpated no return of the disease at that particular spot occurred. If the tumour was only partially removed return was certain. Microscopically the structure of the tumours was that of granulomata, and in the new formation a mycelial structure could be demonstrated. Rénon was not able to determine positively the nature of the parasite, but thinks that the case was probably one of aspergillar mycosis.

As to infections by *Blastomycetes* or yeast fungi, many new facts have accumulated during recent years. And in this class of diseases, again, the formation of granulomata is of extremely common occur-

rence. Yeasts infective for man have been described by Gilchrist, by
Busse, and by Curtis. A large number of yeasts have been found
experimentally to have marked pathogenic properties for mice, guinea-
pigs, and rabbits. In guinea-pigs and rabbits, when the progress of
the disease is not too acute to permit of their development, charac-
teristic tubercles or granulomata containing the specific fungi, are
found in various parts. Mafucci and Sirleo have described two cases
of natural disease in guinea-pigs produced by yeast infection, and
Professor Tokishige, in his paper *Ueber Pathogene Blastomyceten*
(*Central. fur. Bakt.*, Feb. 1896), has carefully described a yeast in-
fection in horses and cattle. This disease is known in Japan as
" worms," and carries off year by year a large number of horses. It
occurs more frequently in the plains than in mountainous districts,
and is more prevalent during wet than during dry years. The
commonest manifestation of the disease is the development of sub-
cutaneous nodes with enlargement of the nearest lymph glands. The
primary nodes nearly always occur in the subcutaneous tissue, but ex-
ceptionally a primary infection of the nasal mucous membrane occurs.
The following figures indicate the usual primary location of the
disease ; the nodes appeared first :—

On an anterior limb in	30	cases.
On the chest (front)	17	"
On the neck	14	"
On the belly	11	"
On a posterior limb	11	"
On the side of the thorax	10	"
On the genital organs	8	"
On the face	6	"

The principal morbid appearances found in horses which had died or
had been slaughtered on account of the disease were circumscribed
nodes in the subcutaneous tissue, varying in size from that of a pea
to that of a walnut, and these were frequently in a softening condition.
In the submucous tissue of the respiratory tract also nodes were some-
times found, and similar formations occurred in the lymph glands,
the testicles, the lungs, and other organs. These nodes, on micro-
scopic examination, proved to be granulomata, and frequently con-
tained giant cells ; in them yeast-like cells were seen both included
in the tissue cells and lying free. From these granulomata Tokishige
isolated a yeast to which he has given the name *Saccharomyces
farciminosus*. This yeast was tested by experimental inoculation on
various animals, but gave only negative results, in spite of which,
however, there can be but little doubt as to the parasite being the
actual cause of the disease. Tokishige saw also four cows which
suffered from a similar affection. All the cows presented numerous
firm, painless, circumscribed nodules in the subcutaneous tissue,
varying in size from that of a hazel nut to that of a walnut. And in
these nodules the yeast fungus was identified microscopally. In none
of the cases in the cows was there any tendency to softening in the
granulomata. Tokishige remarks on the resemblance of the con-
dition which he describes to the disease of horses known in Italy and
France as lymphangitis epizootica, " African farcy," and " Neapolitan
farcy," and he further suggests that it is identical with the "cattle farcy"
occurring in the West Indian Islands, and especially in Guadaloupe.

The disease known as lymphangitis epizootica, and popularly as "mal del verme," and by the other local names first mentioned, has been particularly observed as affecting horses and mules in France, Italy, Algeria, Egypt, and Guadaloupe, but has never been recorded as occurring in this country. According to Nocard this disease often commences as a lymphangitis in connection with some accidental pre-existing wound, but sometimes independently of any previous lesion. The lymphangitis may remain localised in one region, or may become widely distributed throughout the subcutaneous tissue. Along the course of the enlarged and hardened lymph vessels small tumours, about the size of a hazel nut or larger, develop. These tumours, at first hard and painless, may eventually break down, and, becoming adherent to the skin, ulcerate; or they may undergo a process of gradual involution, and finally disappear. The primary lesion may appear in the nasal mucous membrane. Infection of the lungs and other viscera is, according to Nocard, very rare. The disease, in whatever country it occurs, would appear to cause a large mortality amongst those attacked. The nodes which occur along the course of the lymphatic vessels are granulomata, and contain in their cells the parasite which causes the disease. As the aspergillar mycosis in cattle sometimes presents clinically marked resemblances to certain forms of tuberculosis, so lymphangitis epizootica has an obvious likeness to the more chronic form of glanders, and in many cases a differential diagnosis could only be established after the 'use of the mallein test, or by examination by bacteriological methods of the products of the disease. The micro-organism causing lymphangitis epizootica was discovered by Rivolta, and named by him *Cryptococcus farciminosus*. The parasite was described as a large oval coccus measuring from 3 to 4 μ in diameter, and surrounded by a distinctly double-contoured refractile membrane. It reproduced itself by budding like a yeast. Fermi and Aruch, who some years later made a careful study of the parasite, came definitely to the conclusion that it really belonged to the blastomycetes. Comparing the disease described as "worms" by Tokishige with the lymphangitis epizootica of Rivolta, we find the following points of difference:—"Worms" is caused by a parasite, *Saccharomyces farciminosus*, which measures 3·7 to 4 μ in length by 2·4 to 3·6 μ in breadth, and which amongst other characteristics has an extremely slow rate of growth on the ordinary laboratory media. Thus on gelatin, agar, or potato there was no obvious development for thirty days. Fermi and Aruch on the contrary obtained a growth of the parasite of lymphangitis epizootica on potato in three days. The diameter of *Cryptococcus farciminosus* is given as being about 4 μ. The prominent hard lymphatic vessels, which are very noticeable in lymphangitis epizootica, are not specially mentioned by Tokishige as a feature of "worms." The inoculation experiments in the case of either parasite seem up to the present to have been alike unsatisfactory. Lymphangitis epizootica has not been recognised as such in any but equine species, whilst "worms" occurs sometimes in bovine as well.

On the whole, therefore, we are justified in assuming that, whilst both diseases are probably due to a blastomycetic parasite, the specific fungus is not the same in each case.

The following case, which I have lately investigated, was probably

one of infection by a yeast parasite, although the evidence as to this is not absolute.   The case is, however, of sufficient importance to be recorded, as no such condition has previously been described in this country, nor, save the examples of granulomata in cows already referred to, elsewhere.

In January of this year I received from Dr Bond, the Medical Officer of Health for Holborn, portions of a carcase of cow-beef which had been seized and condemned as unfit for human food. The following general description of the carcase was supplied to me by Dr Bond. " The meat contained a large number of yellowish tubercles, which were distributed over the whole carcase, but were especially numerous on the outsides of the buttocks, the shoulders and the extremities.   All the tubercles were situated within 3 inches of the surface of the muscles, and many were obvious on the surface.   No trace of the disease was to be seen on the pleural surface of the thorax or within the abdominal cavity.   The meat itself appeared, except for the tubercles, to be in a sound condition.   There had obviously been much wasting of the subcutaneous fat during life." The cow had been sent up from Sussex, and was slaughtered in the Islington Cattle Market.   No particulars of the life history of the animal could be obtained.   Dr Bond sent me two large pieces of muscle from the rump, which showed a number of the nodules or tubercles which he had noticed.   These nodules formed round yellowish-white masses, either immediately below the muscle sheath and showing on the surface, or else entirely in the muscle substance and just below the fascia.   No nodules were to be seen at a greater depth than 1 inch from the surface in the specimens which I received. The nodules could be readily shelled out from the muscle surrounding them, bringing away on their surface just a trace of muscle fibre here and there.   The nodules were very hard, and on cutting gave a sensation as when cartilage is being cut.   They were nearly spherical in shape, and in diameter varied from ·2 cm. to ·8 cm.   They were scattered about at irregular intervals ; in some places there were clusters of them quite close together, in other places there were areas of muscle quite free from them.   Under the microscope the forma-tions showed the general characters of granulomata, and all examined were limited by a definite capsule of close fibroid tissue.   Around some of the nodules there was a slight indication of small round-celled infiltration in the muscle fibre, which latter appeared otherwise perfectly natural.   No giant cells were found in a large number of sections examined, nor did any of the nodules show signs of caseous degeneration either macroscopically or microscopically.   In many of the sections, and especially in those stained by Gram's method, small oval bodies were seen lying within some of the cells.   The cells con-taining these bodies were mostly situated at the periphery of the nodule and close to the fibrous capsule.   These intracellular bodies were for the most part distinctly oval in shape, and now and again specimens could be seen which appeared to be surrounded by a narrow refractile zone.   The· bodies in question took the anilin gentian violet stain, used according to Gram's method, very deeply, and retained it after prolonged washing in alcohol.   No cells in the act of budding were seen in the stained sections.   In fresh unstained preparations, however, appearances more characteristic of yeast cells

could be seen. After teasing out small portions of a nodule in glycerine and water small double-contoured bodies could be seen, both lying free and enclosed within larger tissue cells. Several pairs of such cells still united were observed, and once or twice three cells, still joined together, were noticed. Many of these cells, when fresh, showed a very distinct refractile capsule. From the experience which I have had of the appearance in the tissues of various known yeasts with which animals have been experimentally infected, I feel practically certain that the bodies in question were of a similar nature. In the fresh teased-out specimens the yeast-like cells appeared to be far more numerous, and were more characteristic than in the hardened and stained sections, an occurrence which quite corresponds with what one notices when examining granulomata produced by the artificial inoculation of pathogenic yeasts. The sections stained by various appropriate methods did not show the presence of any cocci or bacilli. Cultivation experiments gave only negative results.

This case, then, I regard as one of yeast infection, and similar to the disease described by Tokishige, with which, so far as the anatomical appearances are concerned, it corresponds closely. Having come to this conclusion, I showed the specimen to Professor M'Fadyean of the Royal Veterinary College, and he informed me that he had never, with the possible exception of one case, seen a similar pathological condition in cattle in this country. The disease may therefore certainly be considered to be uncommon. The possible exception to which Professor M'Fadyean referred was a case from which a specimen is in the museum of the Royal Veterinary College. The specimen consists of a cow's tail and a separate portion of muscle, the latter probably from the rump. Along the surface of the caudal muscles are arranged a series of hard, oval, whitish nodules, varying in size from that of a filbert nut to that of a walnut. In the portion of muscle preserved are similar nodules. In the central portion of some of these nodules there is well-marked caseous degeneration. Professor M'Fadyean was kind enough to allow me to take one of the nodules in the muscle for further examination. Microscopic examination showed the structural details characteristic of a granuloma. There were no giant cells to be seen, and the fibroid capsule surrounding the nodule was not quite so well defined and compact as in my case, but the state of the latter would depend of course to a certain degree on the age of the nodule. I was not able to identify in the sections any of the intracellular bodies which in the former case were described as yeast cells. The prolonged hardening in spirit which the specimen had undergone would, however, render the chance of identifying yeast cells in any case a matter of great difficulty; for experimental work has shown that, using such staining methods as we at present have at our disposal, the identification of yeast cells in sections of animal tissue is very greatly affected by the hardening process which has to be gone through.

These two cases are of interest as representing disease in cows of a nature which, whether it be of frequent occurrence or not, has not hitherto been recognised as occurring in this country. In my own case, I am strongly of opinion, in spite of the absence of confirmatory cultivation experiments, that we have to deal with an infective disease caused by a parasite belonging to the *Blastomycetes*. The

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specimen in the museum evidently represents also a case of infective disease with the formation of granulomata. In this case one cannot say what the nature of the infective agent was, but one may say with some certainty that it was not any of the parasites which have been recognised as causing the formation of granulomata in cows in this country.

Amongst the infective diseases of horses and cattle which are characterised by the formation of granulomata must be included the "Bursattee" of the East Indies, an affection which has only been recorded in connection with the former animals, and the "Leeches" of certain parts of the United States, an apparently similar disease from which both species suffer. No such disease is known in animals in this country; one has therefore to rely entirely on the descriptions of those writers who have studied "Bursattee" and "Leeches" in the localities in which they occur. Dr Pierre Fish in the *Twelfth and Thirteenth Annual Reports of the Bureau of Animal Industry*, U.S.A., 1895-96 gives a very interesting historical account of these diseases, which are he considers probably identical in their nature. The first account of "Bursattee" was given by Vet.-Surgeon Kerr, in a short note "On the Disease to which Horses are Subject in the East Indies, termed Bausette" (*The Veterinarian*, 1829, Vol. II.) It is stated that the disease "first makes appearance in the form of small tumours, generally about the angles of the lips, or in the face or scrotum. These, if let alone, suppurate and become a bausette ulcer, which from exposure to air rapidly spreads and becomes scirrhous." Mr Kerr adds that slight abrasions often become infected, that horses alone are subject, that usually the horse suffers only locally, and finally that the disease has some connection with the rainy season. More detailed accounts of the disease are given by, amongst others, F. Smith, M.R.C.V.S. ("Bursattee," *The Veterinary Journal*, 1879, Vol. IX.) and George A. Oliphant, F.R.C.V.S. ("Bursati," *The Veterinary Journal*, 1880, Vol. XI.) The former writer notes that "in all cases the disease originates in the subcutaneous tissue." The tumours formed are called "kunkur stones," and the evolution of them is as follows: "The commencement of a sore on the lip (where in consequence of thinness of the skin we are most satisfactorily able to trace the process) is marked, first by swelling of the part, with heat and pain on pressure. In about four to six days it is possible to detect that one part is becoming much harder than the rest. This circumscribed condition is about the size of a small nut, and by manipulation we may determine that it is situated in the subcutaneous muscular tissue of the part, and increases in size until it is about as large as a shilling." The nodule may become adherent to the skin, and then ulceration occurs; "on examining the sore externally to the ulcerating surface the intense hardness of the tumour is very perceptible (so that it feels like a stone under the fingers)." Mr Smith inoculated himself, eleven natives, who volunteered for the experiment, several horses, and a dog with the "kunkur matter," but with negative results in every case. In this paper Mr Smith suggests that the disease is probably due to a vegetable parasite, and remarks that its appearance is greatly influenced by the season of the year. Mr Oliphant's paper supplements that last quoted in several important details, and especially as to the

anatomical characters of the disease. The bursattee nodule is thus described : " To the eye it is very like tubercle, occurs in more or less nodular masses of various dimensions, from the size of a millet seed to that of a small bean or even larger . . . when fresh it cuts like cartilage and when dried it is distinctly gritty. In all these points it is very like tubercle, but I have failed so far to trace in it one peculiarity of that disease product, softening. . . . The tubercle-like matter will be found frequently thickly studding the substance of the lungs, liver, and spleen, and probably other internal organs and structures, but in no case have I ever seen any attempt at discharge, by ulceration or by softening, from any of these organs." After reading, with the help of the very full bibliography which Dr Fish has compiled, the descriptions of this disease as given by various British veterinary surgeons who have met with it in India, one cannot but be struck by the very close resemblance which it bears to " worms " as described by Tokishige. As to the exact pathology of " Bursattee" one is not able to say very much from the descriptions available. One may, however, with some confidence say that it is not a " cancer," as some writers have described it. Mr Smith in a paper of later date (" The Pathology of Bursattee," *The Veterinary Journal*, 1834, Vol. XIX.) states that " the actual exciting cause of ' bursattee ' is a mould-fungus." This fungus was frequently found in fresh specimens, never in hardened ones. A drawing of the supposed fungus showing a mass of reticulated mycelium is given. But I think that further evidence than is brought forward will be required before the matter can be looked upon as finally settled. In this paper Mr Smith also states that he has been able to reproduce the disease by inoculation with portions of the tumours.

Dr Fish commences the report already mentioned with a long and detailed historical account of " Bursattee," and insists on the resemblance of the disease to " Leeches," which latter he explains is the local name of a disease which is of frequent occurrence amongst horses in Florida. It is also known under other names in various parts of the United States as occurring in horses and cattle, and is not to be confounded with the liver disease in the latter caused by *Fasciola hepatica*, which is also sometimes known as " Leeches." Dr Fish refers very briefly to the clinical history of " Leeches," but one infers that it is identical with that of " Bursattee." As to the pathology, apparently no opportunity occurred of examining fresh specimens either bacteriologically or microscopically, but Dr Fish gives a very careful and minute description of specimens variously prepared. The nodules are obviously granulomata, and amongst the cells Dr Fish believed that he could demonstrate a mycelial structure. Dr Fish comes to the conclusion that he has " demonstrated for the first time the presence of a fungus in ' leeched ' tissues. The fungus is located in the inflammatory growth or nodule, and in some rare instances may be seen ramifying for some short distance into the adjacent tissue." Drawings are given of the alleged fungus, which would appear to resemble in general characters the mould which Smith described as being present in the " Bursattee" nodule. I do not think, however, that either writer has quite proved his point in this particular detail, and further results of Dr Fish's investigations will be awaited with great interest. Meanwhile it

seems to me that a strong reason for accepting with caution Dr Fish's conclusion as to the exact nature of the parasite is furnished by the method which was employed to demonstrate the fungus in the tissues. "As the result of the treatment of the nodules with the 10 per cent. cold solution of caustic potash a very profuse and intricately branched fungus became apparent." I would venture to suggest that this rather severe treatment of the tissues is one which might lead to fallacies, and that, as in the case of "Bursattee," the exact nature of the parasite causing the disease cannot at present be taken as definitely determined.

A REPORT UPON 500 CONSECUTIVE ADMINISTRA-TIONS OF CHLOROFORM TO DOGS, AND 120 TO CATS, WITH THE AID OF AN INHALER.

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IN the *Journal of Comparative Pathology and Therapeutics* for December 1895 there were published some "Notes on the Admini-stration of Chloroform to the Cat and Dog," and in the *Veterinary Record* for 25th December 1897, a further article entitled "A Report upon 250 Consecutive Administrations of Chloroform to Canine Patients." These cases have almost all occurred in the College Free Clinique and been anæsthetised by students of Classes C and D, under, of course, supervision. For assistance in this supervision I am particularly indebted to Messrs Browning and Hall, M.R.C.V.S., College tutors. The animals have been of all ages, breeds, and sizes, and not generally in any way prepared, being placed on the operating table in the usual way, and the operations performed at various intervals after anæsthesia had been produced.

As will be seen on glancing through the columns the operations have been, in some cases, very serious ones.

The inhaler used in the undermentioned cases, with the exception of numbers 97, 107, 110, 119, 125, 127, 131, and 134 in the canine notes, has been the first pattern of those figured in the *Veterinary Record* for 2nd May 1896; in the above numbers the second pattern was used. The dates refer to 1897 and 1898. The 500 consecutive cases are made up by 190 here recorded, 250 in the *Veterinary Record* as stated, and 60 out of the list published in the *Journal of Comparative Pathology and Therapeutics* for December 1895.

[TABLE.

CANINE PATIENTS.

Date.	No.	Breed.	Sex.	Age.	Condition.	Operation.	Time before Anæsthetised.	Time kept under.
June 21	1	St Bernard	F	9 yrs.	Very fat	Removal tumours	4 mins.	1 hour.
,, 23	2	Collie	M	Aged	Good	Removal anal tumours	6 mins.	48 mins.
,, 23	3	Fox terrier	F	2 yrs.	Good	—	2 mins.	4 mins.
,, 25	4	Irish terrier	F	5 yrs.	Good	Operation for hæmatoma	3 mins.	20 mins.
,, 28	5	Irish terrier	M	—	Good	—	3 mins.	23 mins.
July 2	6	Greyhound	M	9 mos.	Good	Reduce dislocation	5 mins.	14 mins.
,, 6	7	Fox terrier	F	3 yrs.	Good	Operation for hæmatoma	8 mins.	31 mins.
,, 9	8	Whippet	M	2 yrs.	Good	Amputation both forelegs	8 mins.	45 mins.
,, 9	9	Maltese terrier	F	Aged	Very fat	Extract tooth	3 mins.	3 mins.
,, 12	10	Bulldog	F	6 yrs.	Good	Curette vagina	3 mins.	12 mins.
,, 16	11	Irish terrier	M	14 yrs.	Fair	—	2 mins.	11 mins.
,, 17	12	Wire-haired terrier	M	2 yrs.	Good	To clip hair	2 mins.	14 mins.
,, 19	13	Maltese terrier	M	Aged	Fair	Reduce dislocation	1 min.	8 mins.
,, 19	14	Fox terrier	F	9 yrs.	Very fat	Oöphorectomy	3 mins.	40 mins.
,, 20	15	Collie	F	16 yrs.	Good	Removal mammary tumours	3 mins.	50 mins.
,, 20	16	Fox terrier	F	3 yrs.	Good	For entropion	8 mins.	17 mins.
,, 20	17	Fox terrier	F	9 yrs.	Very fat	To irrigate abdomen	2 mins.	28 mins.
,, 21	18	Retriever	M	2 mos.	Very weak	—	1 min.	8 mins.
,, 22	19	Wire-haired terrier	M	2 yrs.	Good	To dress skin	3 mins.	15 mins.
,, 22	20	Fox terrier	M	Aged	Fair	Extract teeth	2 mins.	6 mins.
,, 23	21	Boarhound	M	3 yrs.	Good	Amputate tail	5 mins.	30 mins.
,, 25	22	Manchester terrier	M	12 mos.	Good	Extract teeth	1½ mins.	9 mins.
,, 25	23	Collie	M	—	Good	Removal ingrowing nails	2 mins.	5 mins.
,, 28	24	Italian greyhound	F	7 yrs.	Good	Amputation of leg	4½ mins.	1 hour.
Aug. 9	25	Retriever	M	7 mos.	Good	—	6 mins.	10 mins.
,, 11	26	Schipperke	M	6 mos.	Poor	Extract teeth and remove tumour	2 mins.	22 mins.
,, 12	27	Wire-haired terrier	M	12 mos.	Good	Examine skin	2 mins.	5 mins.
,, 14	28	Retriever	M	18 mos.	Good	For entropion	3 mins.	15 mins.
,, 20	29	Fox terrier	M	—	—	—	1 min.	3 mins.
,, 22	30	Fox terrier	F	6 mos.	Poor	Oöphorectomy	1½ mins.	22 mins.
,, 23	31	Spaniel	M	—	Very fat	Remove tumour	1 min.	23 mins.
Sept. 2	32	Manchester terrier	—	—	—	—	½ min.	3 mins.
,, 2	33	Mongrel	—	—	Good	—	1½ mins.	5 mins.
,, 16	34	Wire-haired terrier	M	14 mos.	Good	Examine skin	2 mins.	2 mins.
,, 24	35	Schipperke	F	6 mos.	Good	Extract teeth	1½ mins.	15 mins.
,, 25	36	Collie	F	Aged	Good	Removal of tumour	...	47 mins.
,, 27	37	Manchester terrier	M	7 mos.	Poor	Chorea	1½ mins.	4 hours.
,, 29	38	Fox terrier	F	5 mos.	Good	—	2 mins.	3 mins.
,, 29	39	Spaniel	M	10 yrs.	—	—	3 mins.	31 mins.
Oct. 2	40	Collie	F	5 mos.	Good	Oöphorectomy	2½ mins.	24 mins.
,, 6	41	Schipperke	M	12 yrs.	Good	Extract teeth	2½ mins.	28 mins.
,, 10	42	Same dog	—	—	—	Take impressions for false set of teeth	1½ mins.	36 mins.
,, 12	43	Same dog	—	—	—	Ditto	2 mins.	37 mins.
,, 12	44	Clumber spaniel	F	—	Good	Examine larynx	7 mins.	4 mins.
,, 14	45	Fox terrier	F	3 yrs.	Weak	Examine skin	2 mins.	7 mins.
,, 14	46	Fox terrier	M	4 yrs.	Good	For cataract	2½ mins.	6 mins.
,, 15	47	Pomeranian	F	Aged	Fat	For tumour of vagina	1½ mins.	13 mins.
,, 15	48	Retriever	F	3 yrs.	Good	Dress skin	2 mins.	22 mins.
,, 15	49	Schipperke	M	12 yrs.	Good	Fit models of false teeth	2 mins.	1 h. 20 m.
,, 16	50	Collie	F	Aged	Fat	—	4½ mins.	37 mins.
,, 16	51	Greyhound	M	6 mos.	Good	For strychnia poisoning	5 mins.	20 mins.
,, 17	52	Schipperke	M	12 yrs.	Good	To insert false teeth	2 mins.	1h. 22 m.
,, 19	53	Greyhound	F	6 mos.	Good	For convulsions	6 mins.	10 mins.
,, 20	54	Clumber spaniel	M	15 yrs.	Good	Examine eyes	3 mins.	8 mins.
,, 21	55	Fox terrier	M	4 yrs.	Good	Examine scald	4 mins.	5 mins.
,, 21	56	Collie	F	Aged	Good	Examine tumours	6 mins.	5 mins.
,, 23	57	Schipperke	M	Aged	Good	Remove and replace false teeth	2½ mins.	32 mins.
,, 25	58	Fox terrier	F	12 yrs.	Fat	Remove tumours	4 mins.	55 mins.
,, 27	59	Schipperke	M	Aged	Good	Re-insert teeth	2 mins.	40 mins.
,, 28	60	Schipperke	M	10 mos.	Good	Umbilical hernia	3 mins.	15 mins.
,, 28	61	Manchester terrier	F	1½ yrs.	Fat	Oöphorectomy	1½ mins.	12 mins.
,, 29	62	Fox terrier	M	4 yrs.	Good	Hæmatoma of ear	5 mins.	35 mins.
Nov. 1	63	Wire-haired terrier	F	10 yrs.	Good	—	5 mins.	5 mins.
,, 3	64	Fox terrier	M	14 yrs.	Bad—ascites	—	4 mins.	10 mins.
,, 3	65	Fox terrier	F	3 yrs.	Good	Hæmatoma of ear	4 mins.	5 mins.
,, 4	66	Fox terrier	F	4 yrs.	Very fat	—	4 mins.	5 mins.
,, 5	67	Fox terrier	F	7 yrs.	Good	Mammary tumours	2 mins.	26 mins.
,, 7	68	Manchester terrier	M	15 mos.	Poor	—	4 mins.	18 mins.
,, 10	69	Terrier	F	4 mos.	Good	Chorea	3 mins.	16 mins.
,, 11	70	Retriever	M	Aged	Fat	Entropion	5 mins.	15 mins.
,, 11	71	Lurcher	M	7 yrs.	Poor	Chorea	3½ mins.	15 mins.
,, 15	72	Fox terrier	M	3 yrs.	Good	Tumours of mammæ	5 mins.	35 mins.
,, 17	73	Schipperke	M	Aged	Good	To take models for false teeth	4 mins.	12 mins.
,, 22	74	Airedale	F	3 yrs.	Good	Reduce fracture	5 mins.	35 mins.
,, 22	75	Collie	M	4 yrs.	Good	—	7 mins.	18 mins.
,, 23	76	Fox terrier	M	7 mos.	Good	—	4 mins.	9 mins.
,, 23	77	Fox terrier	F	5 mos.	Good	—	1 min.	10 mins.
,, 24	78	Wire-haired terrier	M	9 mos.	Good	—	2 mins.	21 mins.

CANINE PATIENTS—*continued.*

Date.	No.	Breed.	Sex.	Age.	Condition.	Operation.	Time before Anæsthetised.	Time kept under.
Nov. 24	79	Boarhound	M	9 mos.	Good	——	7 mins.	9 mins.
,, 25	80	Scotch terrier	F	7 mos.	Very fat	Umbilical hernia	2½ mins.	18 mins.
,, 25	81	Wire-haired terrier	M	18 mos.	Good	——	4 mins.	6 mins.
,, 26	82	Fox terrier	F	2 yrs.	Good	——	3 mins.	5 mins.
,, 26	83	Manchester terrier	F	7 mos.	Good	——	3 mins.	17 mins.
,, 27	84	Beagle	F	3 yrs.	Good	——	3 mins.	11 mins.
,, 27	85	Wire-haired terrier	F	3 yrs.	Good	——	2 mins.	10 mins.
,, 29	86	Setter	F	10 mos.	Good	——	4 mins.	4 mins.
,, 29	87	Manchester terrier	M	8 mos.	Good	——	4 mins.	15 mins.
Dec. 3	88	Setter	M	2 yrs.	Good	Tumour on penis	5 mins.	45 mins.
,, 10	89	Schipperke	M	Aged	Good	Re-insert false teeth	2½ mins.	22 mins.
,, 11	90	Irish terrier	M	2 yrs.	Good	Extract teeth	3 mins.	2 mins.
,, 15	91	Collie	F	5 yrs.	Good	Removal mammary tumour	6 mins.	25 mins.
,, 15	92	Lurcher	M	Aged	Good	Remove tumour	2 mins.	4 mins.
,, 18	93	Fox terrier	F	2 yrs.	Good	Operation on eyes	3 mins.	7 mins.
,, 22	94	Beagle	M	7 yrs.	Asthmatical	To examine	3 mins.	7 mins.
Jan. 12	95	Maltese terrier	F	9 yrs.	Fat	Mammary tumour	5 mins.	24 mins.
,, 12	96	Irish terrier	M	8 mos.	Good	——	3 mins.	6 mins.
,, 17	97	Fox terrier	M	2 mos.	Very fat	——	2½ mins.	3 mins.
,, 18	98	Fox terrier	F	5 mos.	Good	Laparo-enterotomy	6 mins.	31 mins.
,, 19	99	Fox terrier	F	2 yrs.	Good	Inguinal hernia	5 mins.	29 mins.
,, 20	100	Fox terrier	F	3 yrs.	Good	Tumours in ear	2 mins.	14 mins.
,, 20	101	Fox terrier	M	3 yrs.	Good	Tumour on head	4 mins.	2 mins.
,, 21	102	Italian greyhound	M	8 mos.	Good	Reduce fracture	2 mins.	3 mins.
,, 21	103	Fox terrier	F	2 yrs.	Very fat	——	5 mins.	7½ mins.
,, 22	104	Dandie Dinmont	F	8 yrs.	Poor	Laparotomy—removal tumour from liver	2 mins.	30 mins.
,, 24	105	Fox terrier	M	Aged	Good	Amputation	7 mins.	8 mins.
,, 24	106	Fox terrier	M	18 mos.	Very fat	——	2½ mins.	5 mins.
,, 25	107	Toy Manchester terrier	M	2½ mos.	Good	——	2½ mins.	4 mins.
,, 26	108	Pug	F	Aged	Very fat	——	5 mins.	21 mins.
,, 27	109	Manchester terrier	M	1½ mos.	Good	——	4 mins.	14 mins.
,, 29	110	Fox terrier	M	5 yrs.	Very fat	——	2 mins.	7 mins.
,, 31	111	Fox terrier	F	15 mos.	Good	——	3 mins.	11 mins.
Feb. 1	112	Fox terrier	F	Aged	Very fat	——	2½ mins.	17 mins.
,, 4	113	Fox terrier	M	2 yrs.	Poor	——	4 mins.	2½ mins.
,, 7	114	Retriever	F	6½ yrs.	Very fat	For mammary tumour	7 mins.	28 mins.
,, 14	115	Collie	M	3 yrs.	Good	Amputation of tail	3½ mins.	22 mins.
,, 14	116	Fox terrier	M	Aged	Poor	——	2½ mins.	3 mins.
,, 21	117	Fox terrier	F	Aged	Good	For shoulder tumour	7½ mins.	33 mins.
,, 21	118	Fox terrier	F	Aged	Very fat	For mammary tumour	2 mins.	40 mins.
March 2	119	Yorkshire terrier	F	11 yrs.	Fat	For mammary tumour	3 mins.	14 mins.
,, 8	120	Toy Manchester terrier	M	18 mos.	Good	Amputation of leg	2 mins.	35 mins.
,, 9	121	Fox terrier	F	Aged	Very fat	For mammary tumour	4 mins.	20 mins.
,, 12	122	Fox terrier	M	2 yrs.	Poor	——	5 mins.	18 mins.
,, 15	123	Manchester terrier	F	Aged	Very fat	For tumour	7 mins.	14 mins.
,, 20	124	Fox terrier	M				3½ mins.	13 mins.
,, 23	125	Pug	M	18 mos.	—	——	3½ mins.	12 mins.
,, 23	126	Collie	M	4 mos.	Good	Castration	4 mins.	7 mins.
,, 28	127	Fox terrier	M	4 yrs.	Fat	Operation on paw	3½ mins.	7½ mins.
April 15	128	Fox terrier	—		Good	Removal membrana	4 mins.	3½ mins.
,, 15	129	Fox terrier	F	Aged	Fat	——	4½ mins.	11 mins.
,, 18	130	Manchester terrier	M	8 yrs.	—	——	4 mins.	8 mins.
,, 21	131	Fox terrier	F	Aged	Very fat	——	8 mins.	5 mins.
,, 25	132	Dandie Dinmont	M	—	Very fat	Removal of tumour	4 mins.	23 mins.
,, 25	133	Fox terrier	—	Aged		——	3 mins.	9 mins.
,, 25	134	Welsh terrier	—	6 mos.	Good	Umbilical hernia	5 mins.	22 mins.
,, 29	135	Toy Manchester terrier	M	5 mos.	Good	Reduce dislocations of elbows	1½ mins.	40 mins.
,, 29	136	Fox terrier	M	1½ yrs.	Good	Remove dew claws	6½ mins.	6 mins.
May 3	137	Spaniel	M	2 yrs.	Good	Umbilical hernia	4 mins.	23 mins.
,, 4	138	Fox terrier	M	—	Good	Remove tumour	5 mins.	34 mins.
,, 11	139	Yorkshire terrier	M	8 yrs.	Poor	Extract teeth	2½ mins.	6 mins.
,, 13	140	Bull terrier	M	8 yrs.	Asthmatical	——	4 mins.	11 mins.
,, 19	141	Irish terrier	M	1 yr.	Poor	Chorea	3 mins.	9 mins.
,, 23	142	Manchester terrier	M	7 yrs.	Good	——	1½ mins.	17 mins.
,, 23	143	Fox terrier	M	18 mos.	Poor	Examine lips	3 mins.	10 mins.
,, 23	144	Fox terrier	M	2 yrs.	Poor	Examine lips	2 mins.	1½ mins.
June 4	145	Schipperke	F	10 mos.	Fat	Oöphorectomy	2 mins.	30 mins.
,, 7	146	Wire-haired terrier	M	18 mos.	Good	——	4½ mins.	15 mins.
,, 9	147	Pug	F	7 yrs.	Fat	Oöphorectomy and curette vagina	4 mins.	34 mins.
,, 11	148	Boarhound	F	18 mos.	Good	——	6 mins.	10 mins.
,, 11	149	Retriever	M	18 mos.	Weak	Urethrotomy	3 mins.	40 mins.
,, 11	150	Retriever	M	Aged	Good	Castration	10 mins.	5 mins.

For the following twenty consecutive cases I am indebted to Mr Anderson, a Class D student, who anæsthetised the patients during the last college vacation, using successfully an inhaler of the first model without any mishap:—

No.	Breed.	Sex.	Age.	Time before anæsthetised.	Time kept under.
151	Irish terrier	M	2 years	7 mins.	10 mins.
152	Fox terrier	M	Aged	8 mins.	7 mins.
153	Manchester terrier	F	3 years	5 mins.	12 mins.
154	Wire-haired terrier	M	—	6 mins.	10 mins.
155	Pug	M	Aged	7 mins.	5 mins.
156	Skye terrier	F	—	4 mins.	19 mins.
157	Wire-haired terrier	M	—	6 mins.	5 mins.
158	Manchester terrier	M	—	5 mins.	12 mins.
159	Setter	M	—	8 mins.	10 mins.
160	Collie	M	—	10 mins.	7 mins.
161	Bull terrier	—	—	7 mins.	10 mins.
162	Fox terrier	M	—	5 mins.	5 mins.
163	Irish terrier	M	—	6 mins.	5 mins.
164	Toy terrier	—	—	4 mins.	10 mins.
165	Mongrel Newfoundland	—	—	10 mins.	15 mins.
166	Fox terrier	F	—	7 mins.	13 mins.
167	King Charles spaniel	M	—	4 mins.	8 mins.
168	Pug	M	—	6 mins.	7 mins.
169	Airedale	F	—	4 mins.	6 mins.
170	Fox terrier	M	—	6 mins.	5 mins.

For the following twenty consecutive cases I am indebted to Mr C. F. Hulford, M.R.C.V.S., of Haslemere, Surrey, who used the first pattern of inhaler:—

Date.	No.	Breed.	Sex.	Age.	Condition.	Operation.	Time before anæsthetised.	Time kept under.
Apl. 20	171	Collie	F	3 years	Good	Remove mammary tumour	12 mins.	36 mins.
„ 24	172	Wire-haired terrier	M	15 mos.	Good	Remove tumour	17 mins.	6 mins.
June 4	173	Fox terrier	M	11 mos.	Fair	Reduce fracture	8 mins.	17 mins.
„ 17	174	Pug	M	18 mos.	Very fat	Explore nostrils	—	5 mins.
„ 21	175	Fox terrier	M	15 mos.	Fair	Reduce fracture	6½ mins.	20 mins.
Aug. 5	176	Fox terrier	F	1 year	Fat	Remove fœtus	5 mins.	35 mins.
„ 9	177	Retriever	M	6 years	Good	Operation on eye	7 mins.	15 mins.
Sept. 8	178	Dachshund	F	3 years	Fat	Reduce fracture	10 mins.	40 mins.
„ 20	179	Lurcher	M	3 years	Fair	Amputation of tail	5½ mins.	8 mins.
Nov. 23	180	Foxhound	M	2 years	Good	Castration	7 mins.	8 mins.
Dec. 6	181	Retriever	M	14 mos.	Good	Suture wounds	3½ mins.	6½ mins.
„ 21	182	Collie	F	7 years	Good	Remove tumours	6 mins.	20 mins.
Feb. 3	183	Retriever	M	3 years	Good	Remove tumours	10 mins.	35 mins.
„ 18	184	Toy terrier	F	8 years	Fair	Extract teeth	2 mins.	7 mins.
Mch. 3	185	Pug	F	10 mos.	Fat	Remove mammary tumour	10 mins.	16 mins.
Apl. 6	186	Newfoundland	M	4 years	Good	Operation on jaw	17 mins.	27 mins.
May 7	187	Poodle	M	2 years	Good	Operation on rectum	11 mins.	19 mins.
„ 18	188	Bull terrier	M	10 mos.	Fat	Remove shots from wound	13 mins.	28 mins.
June 6	189	Italian Greyhound	F	6 mos.	Thin	Reduce fracture	2½ mins.	85 mins.
„ 16	190	Spaniel	M	15 mos.	Good	Reduce fracture	8 mins.	27 mins.

Out of the whole 500 administrations by one or other patterns of this inhaler the only death that occurred was that of an aged fat pug (No. 67 in the first list appearing in the *Record* for 25th September

1897), and this was fully accounted for on *post-mortem* examination by the discovery of an extensive rupture of the portal vein, the abdomen being full of blood. Of the 190 cases here mentioned the only ones which gave any cause for anxiety were numbers 80 and 144. In Case 80 the animal was a Scotch terrier, seven months old and very fat ; it was operated upon for umbilical hernia and placed on the operating table on its back. Respirations suddenly ceased, but recommenced after the administration of 3 minims of hydrocyanic acid (Scheele's) and artificial respiration, the animal making a good recovery.

In Case 144 the animal, a fox terrier about eighteen months old, had not been under the anæsthetic more than two minutes before respiration suddenly ceased. Artificial respiration and 2 minims of hydrocyanic acid, together with the application of ammonia vapour to the nostrils, soon brought the animal out of danger. I think in this case overdosage was clearly indicated, as the animal was in very poor condition and the bellows of the inhaler were carelessly and rapidly worked the whole time, 1¼ drachms of chloroform being used in the three-and-a-half minutes.

FELINE PATIENTS.

Date.	No.	Sex.	Age.	Condition.	Operation.	Time before Anæsthetised.	Time kept under.	Amount Chloroform used.
Mch. 18	1	M	—	Good	For Röntgen's Rays	2 mins.	3½ mins.	—
,, 18	2	Same	Cat	—	For Röntgen's Rays	2 mins.	2½ mins.	—
,, 19	3	M	—	Good	Amputation of foreleg	8 mins.	32 mins.	1 dr. 45 m.
,, 20	4	M	3 years	Poor	Castration	2 mins.	9 mins.	—
,, 24	5	M	—	Good	To examine wound	—	5 mins.	—
,, 24	6	F	—	Good	Lance abscess	1½ mins.	5 mins.	—
April 17	7	F	2 years	Good	Examine skin	2 mins.	5 mins.	—
,, 18	8	M	—	Good	To examine	3 mins.	15 mins.	1 dr.
June 6	9	F	18 months	Fair	—	3 mins.	9 mins.	40 m.
Aug. 13	10	M	2 years	Good	Excision membrana nictitans	5 mins.	2 mins.	—
,, 24	11	F	3 years	Good	—	1¾ mins.	2¾ mins.	—
Nov. 14	12	F	18 months	Paralysed	To examine hind parts	1¼ mins.	15 mins.	1 dr. 50 m.
,, 14	13	M	4 years	Good	—	2 mins.	12 mins.	1 dr.
Dec. 2	14	F	2 years	Pregnant	—	2 mins.	11 mins.	2 dr.
,, 19	15	F	3 years	Good	—	1¼ mins.	6½ mins.	—
Feb. 8	16	F	18 months	Poor	Amputation of rectum	3 mins.	42 mins.	1 dr. 40 m.
May 10	17	F	2 years	Good	—	2 mins.	8 mins.	—
,, 25	18	F	—	—	—	3 mins.	Few mins.	—
June 15	19	F	3 months	Good	Oöphorectomy	2 mins.	10 mins.	—
,, 25	20	M	3 years	Good	—	1 min.	7 mins.	—
July 2	21	F	Aged	Poor	—	1 min.	15 mins.	—
,, 2	22	F	11 years	Good	—	1¼ mins.	19 mins.	—
,, 7	23	M	Aged	Good	—	1 min.	12 mins.	—
,, 7	24	F	2¼ months	Good	—	1 min.	12 mins.	—
,, 19	25	—	4 years	Good	Remove cyst	1 min.	13 mins.	—
,, 22	26	F	—	Good	Hæmatoma of ear	1 min.	15 mins.	2 dr.
,, 23	27	F	—	Good	Remove membrana	1¼ mins.	1 min.	—
,, 23	28	F	2 years	Pregnant	Hysterectomy	1 min.	40 mins.	4 dr. 50 m.
,, 29	29	M	5 years	Good	Castration	2 mins.	18 mins.	1¼ dr.
,, 30	30	F	3 months	Good	Oöphorectomy	½ min.	19 mins.	1 dr.
Aug. 5	31	M	—	—	—	2 mins.	1 min.	—
,, 11	32	M	4 years	Good	Examine skin	1 min.	33 mins.	—
,, 12	33	F	2 years	Good	Examine leg	1¼ mins.	10 mins.	—
,, 18	34	—	—	Good	—	—	8 mins.	—
Sept. 17	35	M	5 years	Good	—	2 mins.	15 mins.	—
,, 23	36	F	—	—	—	1 min.	10 mins.	—
,, 24	37	M	3 years	Good	—	2 mins.	20 mins.	—
,, 27	38	F	2 months	Good	Remove eyeball	8 mins.	16 mins.	1¼ dr.
,, 27	39	M	4 years	Very weak	Laporotomy	2¼ mins.	—	*See Notes.*
Oct. 4	40	M	2 years	—	—	2 mins.	12 mins.	—
,, 7	41	F	6 weeks	Paralysed	To examine quarters	1¼ mins.	12 mins.	—
,, 14	42	M	—	Good	To dress skin	2 mins.	30 mins.	—
,, 16	43	M	8 weeks	Good	—	1 min.	15 mins.	—
,, 16	44	F	5 years	Good	—	2 mins.	11 mins.	—
,, 16	45	M	4 years	Good	—	1¼ mins.	43 mins.	—

FELINE PATIENTS—*continued.*

Date.	No.	Sex.	Age.	Condition.	Operation.	Time before Anæsthetised.	Time kept under.	Amount Chloroform used.
Oct 18	46	M	3 years	Good	Amputation of foreleg	1 min.	38 mins.	1½ dr.
„ 23	47	M	4 years	Good	—	3 mins.	5 mins.	—
„ 23	48	F	2 years	Good	—	1 min.	9 mins.	—
„ 29	49	M	18 months	Good	—	1 min.	8 mins.	—
Nov. 1	50	F	8 months	Good	—	1½ mins.	9 mins.	—
„ 16	51	M	4 years	Good	Operation on thigh	1½ mins.	20½ mins.	—
„ 22	52	F	4 years	Good	—	2 mins.	9 mins.	—
„ 22	53	M	Aged	Very fat	—	3 mins.	1 hr. 50 mins.	—
„ 23	54	F	3 years	Good	Oöphorectomy	3 mins.	30 mins.	1 dr.
„ 23	55	M	3 years	Good	Pass probang	1½ mins.	—	—
Dec. 14	56	M	4 years	Weak	Puncture bladder	1 min.	11 mins.	—
„ 15	57	F	3 years	Good	Lance abscess	1½ mins.	5 mins.	--
„ 15	58	M	4 months	Good	Castration	2½ mins.	5 mins.	52 m.
„ 15	59	M	6 months	Good	Castration	1 min.	1½ mins.	—
„ 16	60	F	8 months	Good	Oöphorectomy	2½ mins.	22 mins.	—
„ 18	61	F	5 months	Good	Lance abscess	—	—	See Notes.
Jan. 17	62	F	2 years	Good	Pass probang	2 mins.	3 mins.	—
„ 18	63	M	12 months	Good	Open sinus	2 mins.	7 mins.	—
„ 25	64	M	3 months	Good	—	1½ mins.	5 mins.	—
Feb. 14	65	F	18 months	Good	Overo-bysterectomy	1½ mins.	33 mins.	—
„ 17	66	M	2 years	Good	Dress skin	3 mins.	25 mins.	—
„ 17	67	F	3 years	Good	—	3½ mins.	30 mins.	—
„ 19	68	M	4 years	Good	—	5 mins.	12 mins.	—
„ 19	69	M	3 years	Good	Lance abscess	1½ mins.	17 mins.	—
„ 21	70	F	2 years	Good	Lance abscess	3½ mins.	25 mins.	—
„ 22	71	F	12 months	Good	Overo-hysterectomy	1 min.	30 mins.	1 dr. 20 m.
„ 22	72	F	2 years	Good	—	1½ mins.	26 mins.	—
„ 22	73	F	3 years	Good	Overo-hysterectomy	1½ mins.	47 mins.	—
„ 26	74	F	3 years	Good	Excise omentum	1½ mins.	14 mins.	—
Mch. 2	75	—	2 years	Good	—	1 min.	16 mins.	—
„ 8	76	F	4 months	Good	Oöphorectomy	2 mins.	20 mins.	—
„ 17	77	M	2 years	Good	—	—	25 mins.	—
„ 20	78	M	—	Fat	—	1½ mins.	18 mins.	—
„ 23	79	M	2 years	Fat	Puncture bladder	2 mins.	11 mins.	—
„ 24	80	Same	Cat	Fat	Puncture bladder	1½ mins.	12 mins.	—
„ 14	81	F	12 months	Good	Amputate tail	2½ mins.	30 mins.	—
„ 15	82	M	Aged	—	—	1½ mins.	15 mins.	—
„ 18	83	M	12 months	Good	Castration	1 min.	10 mins.	—
„ 19	84	M	5 years	Good	Amputate tail	1 min.	21 mins.	—
„ 21	85	M	2 years	Good	Castration	1½ mins.	8½ mins.	—
April 21	86	M	1 year	Good	Castration	1½ mins.	4½ mins.	—
„ 22	87	F	2 years	Good	Oöphorectomy	1½ mins.	33 mins.	—
„ 25	88	F	1½ years	Good	Oöphorectomy	1½ mins.	21 mins.	—
„ 25	89	M	—	Good	Operation on leg	1½ mins.	9 mins.	—
„ 26	90	—	—	Good	—	1 min.	24 mins.	—
„ 27	91	—	—	Good	Oöphorectomy	1½ mins.	24 mins.	—
May 10	92	M	2 years	Good	—	2 mins.	13 mins.	—
„ 11	93	M	3 years	Good	Extract and scale teeth	1 min.	8 mins.	—
„ 16	94	M	2 years	Good	—	1½ mins.	9 mins.	—
„ 16	95	M	3 years	Good	Hæmatoma of ear	2 mins.	21 mins.	—
„ 19	96	F	18 months	Very poor	Laporotomy	1½ mins.	—	See Notes.
„ 20	97	F	15 months	Good	Oöphorectomy	1½ mins.	19 mins.	--
„ 27	98	M	Aged	Very fat	Laporotomy	2 mins.	24 mins.	—
„ 27	99	M	3 years	Thin	Laporo-enterotomy	2 mins.	42 mins.	—
June 2	100	F	2 years	Good	Oöphorectomy	1½ mins.	25 mins.	—
„ 3	101	F	11 years	Good	Remove needle from throat	1½ mins.	3 mins.	—
„ 3	102	F	9 months	Good	Oöphorectomy	3 mins.	10 mins.	—
„ 4	103	F	10 months	Good	—	2 mins.	11 mins.	—
„ 9	104	F	11 months	Good	Oöphorectomy	2 mins.	16 mins.	—
„ 9	105	M	3 years	Good	—	1 min.	13 mins.	—
„ 10	106	F	2 years	Good	Oöphorectomy	1 min.	34 mins.	—
„ 10	107	F	9 months	Good	Oöphorectomy	1 min.	22 mins.	—
„ 13	108	M	Aged	Good	—	2 mins.	15 mins.	—
„ 13	109	F	—	Good	Pass probang	4 mins.	2 mins.	—
„ 13	110	M	12 months	Good	—	55 secs.	—	See Notes.

The dates here mentioned refer to 1896, 1897 and 1898.

Ten consecutive cases (Nos. 7 to 16 inclusive) of those recorded in the *Journal of Comparative Pathology and Therapeutics* for December 1895 belong to this series, thus making a total of 120 in all anæsthetised by this pattern of inhaler.

Out of these 120 administrations three deaths occurred, and in one other case resuscitative efforts had to be made.

CASE 39.—The animal, which was very weak and in poor condition, was suffering from hemorrhagic cystitis, the suspected cause being calculi or sabulous material in the neck of the bladder. An operation was about to be performed to relieve this. After being under the

anæsthetic for two minutes the respirations suddenly ceased. The hobbles were immediately loosened, 3 minims of hydrocyanic acid applied to the back of the tongue, and artificial respiration resorted to. Thirty seconds later respirations recommenced, at first feebly, then becoming rapidly strong, and in two minutes the animal was out of danger.

CASE 59 was an animal suffering from an exceedingly large and painful abscess in the left axilla. A few whiffs of chloroform vapour were given, but before the animal had had time to become anæsthetised it was noticed that respiration had ceased. Unfortunately no medicinal antidotes were at hand, and, as it was thought that probably the cat was only voluntarily holding its breath, death had occurred some few moments before artificial respiration was applied ; all subsequent efforts at resuscitation failed. Unfortunately a *post-mortem* examination was not made.

CASE 96 was a cat in very poor condition being chloroformed for laparotomy. Anæsthesia was obtained in one-and-a-half minutes, but about three minutes later respiration suddenly ceased. Hydrocyanic acid, ammonia vapour, and artificial respiration were all applied as quickly as possible but, although the animal gave a few gasps, efforts at resuscitation failed. Here again, unfortunately, no *post-mortem* was made.

CASE 110.—This animal went under the chloroform in fifty-five seconds, and unfortunately the student who was anæsthetising kept forcing the bellows as hard as possible during the next four minutes. Respirations suddenly ceased, no resuscitative measures were adopted, and the patient died.

The conclusions to be drawn from the above cases simply confirm those which have been made before, viz. :—

1. That, if proper precautions be taken, the dog is a good subject for chloroform. These proper precautions especially refer to the way in which the anæsthetic is administered and the method of restraint. For successful administration chloroform must be given slowly, steadily, and gradually ; and, as regards restraint, the abdominal position on the operating table, with the legs spread just so wide apart as to prevent struggling, is the safest, as it allows free play for the organs in the chest.

2. That with the cat much greater care must be taken than with the dog, as this animal is much more susceptible to the influence of the anæsthetic.

3. That the anæsthetist should do nothing else but attend to his own work, particularly noticing the respirations, and not be watching the operation.

4. That young animals are more susceptible and require more care than old ones.

5. That antidotes should be always at hand and ready for application, if necessary, upon the slightest signs of danger being shown.

CEREBRAL TETANUS AND IMMUNITY AGAINST TETANUS.

By MM. E. ROUX and A. BORREL.[1]

I.

TETANUS has always been considered as a disease of the nervous system, but it is only since the discovery of the tetanus toxin that one can say with more precision that tetanus is a poisoning of certain nerve cells. The tetanus poison acts on the cells of the spinal cord, the lesion of which determines the characteristic contractions ; when injected under the skin or into the blood the poison proceeds to attack these cells by preference.

There exists, in fact, a veritable affinity between the nerve cells and the tetanus toxin. This affinity is manifested in the following experiment, which was suggested by that of MM. Wassermann and Takaki.[2] Some of the substance of the cerebrum of a guinea-pig is triturated with some tetanus toxin ; when submitted to the action of a centrifugal machine the mixture separates into two strata, namely, one of nervous matter at the bottom of the vessel, and above that an opaline liquid.[3] If the proportion of cerebrum and toxin are properly chosen one observes that the liquid contains hardly any of the tetanus poison. The latter is fixed by the nervous tissue and deposited along with it. It has not been destroyed as M. Wassermann believes ; it adheres to the débris of the cerebral substance after the manner of a colouring matter, and, as M. Metchnikoff[4] has shown, may again be put in evidence. The toxin has changed its state, but its nature has not been modified.

The real interest of M. Wassermann's experiment is that it demonstrates *in vitro*, and, so to speak, in a tangible fashion, this affinity between the nerve cells and the tetanus toxin.

What transpires in our test-tube takes place also in the body. The tetanus toxin when injected under the skin of the hind foot of a guinea-pig will be fixed by the cells of the spinal cord after a certain number of hours, at the end of which the contractions will appear. The poison arrives at the nervous axis by two ways—one part, according to M. Marie, follows directly the track of the nerves, and that is why in animals the rigidity always commences in the region where the injection has been practised ; another part of the poison penetrates into the blood, whence it is extracted by the nerve cells, and perhaps also by other cells according to their affinity.

The specific affinity of the nervous elements for the tetanus toxin comes into play when one introduces a little of the poison into the substance of the cerebrum of a rabbit. One thus determines a characteristic disease—cerebral tetanus. This fact will suffice to overthrow the opinion of M. Wassermann regarding the existence of a tetanus antitoxin in the normal brain. How can one admit the

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[1] Translated from the "Annales de l'Institut Pasteur," April 1898. A resumé of this article was communicated to the National Congress for Hygiene and Demography at Madrid by M. Borrel on the 12th April 1898.

[2] "Berliner Klinische Wochenschrift," 1898, No. 1.

[3] V. Knorr "Münchener Medicinische Wochenschrift," 1898, No. 12, p. 187.

[4] "Annales de l'Institut Pasteur," 1896, p. 257.

existence of this antitoxin, which does not act even in the place
where it is supposed to be produced? In reality, the mixture of
crushed cerebrum and tetanus toxin is inoffensive because the poison
adheres to the nervous matter, and because when introduced in this
state under the skin of animals it does not diffuse, but is taken in by
the phagocytes and digested in their interior at the same time as the
débris of nerve tissue which serves to support it. It is like the case
of cholera toxin contained in the bodies of the vibrios, as has been
shown by MM. Metchnikoff, Roux, and Salimbeni.

The almost immediate fixation of the tetanus poison on the
nervous elements makes it possible to limit the action of the poison
to a definite group of cells by carrying it directly into contact with
them. One thus obtains a disease, the symptoms of which depend
upon the functions of the intoxicated area.

Thus, when the toxin is injected into the middle of the brain it
provokes in the rabbit and guinea-pig a disease characterised by
extraordinary excitement, intermittent convulsive attacks, motor
disturbance, and polyuria, and which in no way resembles ordinary
tetanus. In these conditions the poison does not act on the spinal
cord, but on the psychical centres and motor regions of the cerebrum,
which react in their own way, so that we see developed an experi-
mental disease with variable symptoms according to the extent of the
region involved.[1]

M. Tizzoni[2] and Mlle. Cattani, after injecting some tetanus toxin
under the dura mater of a rabbit, observed manifestations of cerebral
tetanus, soon followed by rigidity of the limbs. In that case the
experimenters had a case of mixed cerebral and spinal tetanus;
they had probably wounded the surface of the cerebrum, for M.
Vaillard[3] and M. Conrad Brunner[4] have introduced some tetanus
toxin into the arachnoid cavity without determining anything beyond
generalised contractions. Certain observations regarding human
tetanus, supervening in consequence of wounds of the head, relate
symptoms, such as facial paralysis, which might be referred to
cerebral tetanus. But this disease, which is so easy to produce
experimentally, is hardly known, although it well deserves profound
study. For several months M. Morax has undertaken the investi-
gation of it at the Institute Pasteur.

The introduction of the tetanus toxin into the cerebrum is the
simplest thing in the world when one makes by means of a drill a
small hole in the cranial bones; a stop on the drill prevents it pene-
trating too far and avoids wounding the dura mater. It is easy to
make the hole at the same place so as to reach the same region of
the encephalon in comparative experiments. One plunges the needle
of the syringe right into the cerebral substance to a depth regulated
by a stop, and one injects one-twentieth to one-tenth of a cubic centi-
metre according to the dilution of the toxin.

It goes without saying that the operation by itself is inoffensive,

[1] We give to this tetanus provoked by the introduction of the toxin into the brain the name
of cerebral tetanus, to distinguish it from cephalic tetanus and from the hydrophobic tetanus
of Rose.

[2] Tizzoni and Mlle. Cattani "Archiv fur experimentale Pathologie," Vol. XXVII.,
p. 439. 1890.

[3] "Annales de l'Institut Pasteur."

[4] Conrad Brunner "Tétanos céphalique experimentale et clinique," 1894. "Beitrage für
klinische Chirurgie."

that the lesion caused by the needle does not cause any accident, and that if one injects in this manner five times the quantity of sterilised water, physiological solution, or toxin heated to 100°, the rabbit does not afterwards develop any symptom.[1]

For some hours afterwards the animal appears quite at its ease and eats with every appearance of health, but after from eight to twelve hours it becomes restless, constantly changes its position, adopts a peculiar attitude, the hind quarters being elevated as if it could not quite sit down. At the same time it appears to be a prey to hallucinations, seeks to hide its head, and, as if in terror, runs round its cage. If taken out it leaps high up on its legs with the mode of progression of a hare. Often at each leap the hind legs are projected forward in advance of the head. The desire to escape is very marked, the rabbit running round the laboratory, taking refuge in corners, and running against obstacles. The emissions of urine are numerous and abundant. Convulsive epileptiform attacks set in more or less frequently, either spontaneously or on the slightest excitement. The animal falls, grinding its teeth, its neck and limbs convulsed, biting the litter of its cage ; then it gets up again and begins to eat, soon to fall down again.

Sometimes the hind feet are separated in an awkward manner, and muscular tremors make the gait hesitating.

The intensity and duration of the disease vary according to the dose of the toxin. With a rather small quantity, such as one-tenth of a cubic centimetre in certain experiments, the convulsive attacks were incessant, and the entire picture was evolved in from twelve to twenty hours. With smaller doses the affection may last three, five, and even eight or fifteen days. The animal has attacks from time to time, and in the interval it appears normal, without anything restless in its attitude ; it becomes more and more emaciated and dies.

Quite small quantities of toxin set up a tetanus from which the animal may recover, and which is characterised by a tendency on the part of the animal to conceal itself, slight and transient epileptiform attacks, and emaciation. Even in this degree cerebral tetanus persists for a long time, a month and sometimes more.

On account of the size of the rabbit's brain it is possible to introduce the toxin at different points of it. When an injection is made into the cerebellum after about three days there sets in paralysis of the hind quarters, and at the same time convulsive attacks. To avoid implicating more than a limited group of cells it is only necessary to employ a very small quantity of toxin, so that the excess does not diffuse. The poisons which have a great affinity for nerve cells and which fix themselves on these provide us with an original method of exploring the various encephalic territories. In animals with the brain more developed and the faculties better differentiated one will doubtless obtain results interesting for physiology.

The rabbit is rather resistant to the tetanus poison introduced under the skin or into the blood ; 2½ cubic centimetres of our toxin introduced into the cellular tissue give to an animal of 2 kilometres a tetanus which is ordinarily fatal in four days ; one-tenth of a cubic centi-

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[1] At the autopsy one does not find any cerebral lesion appreciable to the eye, and frequently the track of the needle is no longer visible.

metre into the brain provokes an intense cerebral tetanus which kills in less than twenty hours. The resistance of the rabbit to the tetanus toxin, when that is injected in the ordinary conditions, is thus not due to a relative insensibility of the nerve centres, but without doubt to the fact that much of the poison introduced does not reach the nerve cells and is destroyed at some place in the body.

The guinea-pig, which, weight for weight, takes tetanus rather more readily than the rabbit when it receives the toxin under the skin, resists much better the intracerebral injection. When to a guinea-pig of 500 grammes one gives one-hundredth of a cubic centimetre of toxin under the skin of a hind foot, the contractions appear at the twelfth hour and death supervenes towards the fiftieth hour. Another guinea-pig of the same weight received the same dose into the brain; the first symptoms showed themselves after forty-eight hours and the disease lasted three days.

Cerebral tetanus of the guinea-pig is quite as characteristic as that of the rabbit. There are no permanent contractions, but convulsive attacks more or less frequently repeated, after which the animal eats and moves freely. The desire to escape is manifested in these animals also; immediately they are taken out of the cage they set off in a straight line with a rapid movement of the legs, as if moved by a spring; they throw themselves against the first obstacle, fall into convulsions, then get up again, and after a time assume their normal appearance. Cerebral tetanus of the guinea-pig also presents all degrees of intensity, and it may be recovered from.

To provoke distinct symptoms of cerebral tetanus in the rat requires more toxin than is necessary to kill by subcutaneous injection. The period of incubation is from forty-eight hours to three days. If the experimenter did not know that he had injected some tetanus poison, he would never recognise tetanus in the disease which he observes. Psychical manifestations predominate, the rat is restful and watchful, without apparent cause it is seized with sudden fright, and runs madly around its cage. If one opens that it bounds on to the floor, seeks refuge in a corner, and then, the attack having passed off, it allows itself to be seized and remains for a time quiet, notwithstanding the excitement. During the attack it seems to obey some internal impulse, and gives one the impression of an animal seized with madness; in watching it one wonders whether some mental affections may not also be produced by the fixation on certain nerve cells of bacterial toxins elaborated at a given moment in the intestine or in some other part of the body. This state may last for several days; the rat ceases to eat and dies much emaciated. Smaller quantities of the tetanus poison entail merely a cachectic state, which terminates in death after a variable interval.

In mice cerebral tetanus is less dramatic. The dose of toxin which when injected under the skin will kill them in from thirty to thirty-six hours, when introduced into the brain appears at the outset to produce no effect; later the animal is stupefied, slow in its movements, and dies emaciated without tetanic contractions.

To sum up, in place of the permanent contractions of ordinary tetanus we see in cerebral tetanus, excitement, epileptiform attacks, polyuria, and motor disturbance, which give to this disease an easily recognised physiognomy.

II.

CEREBRAL TETANUS AND PASSIVE IMMUNITY.

Injection into the brain is a means of exploring the sensibility of nerve cells to the tetanus toxin, and of ascertaining what becomes of this sensibility in animals immunised in various ways against tetanus.

An animal which has received some antitetanic serum acquires the immunity termed passive, and resists several times the fatal dose of toxin injected under the skin, into its muscles, or into its veins. Is that because its nerve cells have become insensible to the poison? To ascertain that let us place the toxin in direct contact with the cells.

To five rabbits of the same weight we inject into the subcutaneous tissue 5 cc., 10 cc., 15 cc., 20 cc. of antitetanic serum.

The animal treated with 5 cc. is tested by hypodermic injection with a dose of toxin five times greater than the fatal dose, and it remains quite well.

Twenty-four hours after the introduction of the serum the four remaining rabbits receive, at the same time as a control subject, one-tenth of a cubic centimetre of tetanus poison into the brain, that is to say, a dose that in the thigh of a fresh rabbit of the same weight does not determine even a local lesion.

The following day the control rabbit is a prey to convulsive attacks. The rabbits that received 10 cc., 15 cc., and 20 cc. of serum are manifestly attacked, and in them the disease goes on developing. The animal that received 20 cc. dies in forty-eight hours, like the control subject. That which received 10 cc. succumbs on the fourth day, and that which received 15 cc. on the sixth day.

Only the rabbit that received 5 cc. remains quite well.

Nevertheless, the blood of the animals which have succumbed is antitoxic; at the hour of death a drop of blood from the rabbit that received 20 cc. sufficed to neutralise ten times the dose of toxin which is fatal for a mouse.

A drop of blood from the rabbit that received 10 cc., withdrawn at the moment of intracerebral injection, is added to the quantity of tetanus poison capable of giving fatal cerebral tetanus; this mixture when introduced into the brain of a fresh rabbit does not produce any effect.

The mixture of toxin and antitoxin is inoffensive for nerve cells, and a trace of blood from these rabbits, which died in the access of cerebral tetanus, would have sufficed to destroy the tetanus poison if it had come into contact with it. A hæmorrhage caused by the puncture will prevent the appearance of the disease. Without doubt that was what had happened in the rabbit that received 5 cc. of serum in the above-mentioned experiment.

When one operates on a certain number of animals there are always some which remain healthy, for one cannot always avoid a small blood extravasation. Indeed, that is produced in every case, and in sections of the brain the microscope shows blood corpuscles in the puncture. One understands that this minute extravasation of antitoxic blood sometimes suffices to neutralise the small quantity of toxin introduced,

especially in rabbits which have had enormous doses of serum. There
is thus a limit to the experiment. In reality, the portion of the poison
which acts is that which is, so to speak, snapped up by the cells. The
disease is thus frequently slower in the rabbits that have received serum,
these animals then comporting themselves like rabbits which have
received only very small doses of toxin.

Experiments sometimes fail with guinea-pigs immunised by serum,
because their brain is more vascular than that of the rabbit, and less
sensitive to the tetanus toxin. In order to succeed it is necessary to
use a sufficient dose of the poison, though one that is inoffensive when
injected under the skin.

All these facts may be observed with the diphtheria toxin. When
injected into the brain that is more quickly fatal, and in smaller doses,
than when introduced under the skin. It determines in twelve hours
paralysis, soon followed by death. The ordinary lesions, namely
congestion of the suprarenal capsules, and exudation of serosity into
the pleural cavities, are encountered in guinea-pigs as the result of
intracerebral injection. The diphtheritic poison has an affinity not
only for the nervous system but also for other organs, which de-
generate under its action.

Rabbits and guinea-pigs to which one has administered anti-
diphtheritic serum resist enormous doses of toxin introduced under
the skin, but they die if one introduces a little of it into the brain.
The disease is then exclusively nervous ; it lasts longer, and at the
autopsy one finds neither congestion of the suprarenal capsules nor
pleural exudation. All the organs except the nerve cells have been
protected by the antitoxin.

On many occasions we have thus produced cerebral tetanus and
diphtheria on rabbits which had forty-eight to ninety-six hours
previously received up to 40 cc. of antitoxic serum, that is to say,
after the antitoxin had had time to be everywhere diffused. Even
in these conditions it does not protect the nerve cells. The latter
have not got the same affinity for the antitoxin as for the toxin.
Thus the tetanus antitoxin when injected into animals remains in
the blood, while the toxin is extracted out of that and fixed by the
nerve elements. The antidote does not arrive in contact with the
poison, and the two substances, however closely approached, do not
encounter each other. The serum is efficacious against the toxin in-
jected under the skin since the major part of that will pass through
the blood, but it is powerless against the poison which has already
reached the nerve elements.

That is why in declared tetanus serum so frequently fails. At the
moment when one employs it a part of the toxin is already adherent
to the nerve cells ; the antitoxin neutralises much of the poison which
is still circulating, but it does not attack that which is fixed to the
elements of the spinal cord. It limits the poisoning. If that is too
far advanced the disease will follow its course, for the toxin will
diffuse from nerve cell to nerve cell in spite of the antidote.

If this is so, it is not in the blood of tetanic patients that it is
necessary to accumulate the antitoxin in order to cure them ; what
is necessary is to put the antitoxin where the toxin is acting, and
to protect the vital portions of the cord before they have been
attacked.

III.

TREATMENT OF DECLARED TETANUS.

It is for experiment to show whether this is so.

To twenty guinea-pigs of from 400 to 450 grammes one injects into a hind foot a dose of tetanus toxin fatal in about seventy hours.

Eighteen hours afterwards all the guinea-pigs have stiffness of the legs. At the twenty-fourth hour they are all tetanic.

The five largest are put aside as control subjects.

The fifteen others are divided into three lots.

Twenty-four hours after the injection of the toxin one guinea-pig of the first lot receives 1 cc. of serum under the skin. To the other four one administers, right into the cerebral substance, four drops of the same serum in each hemisphere, that is to say, almost a quarter of a cubic centimetre.

One acts in the same way with the guinea-pigs of the second and third lots, which are treated at the twenty-eighth and the thirty-second hour.

The results are as follows :—

The five control subjects succumb at from the sixty-seventh to the seventy-fourth hour.

The three guinea-pigs treated with the serum under the skin die from the sixty-fourth to the seventy-second hour.

The twelve guinea-pigs treated with serum into the brain have their tetanus delayed. The contractions remain limited to one limb, or to the two hind limbs, according to the hour of intervention. A month afterwards the guinea-pigs are quite well, but the contractions still persist.

Out of forty-five guinea-pigs treated at various times in different experiments, thirty-five have survived the intracerebral injection.

Out of seventeen other guinea-pigs which received the serum under the skin in much larger doses,[1] two only have remained alive. Seventeen control guinea-pigs to which no serum had been given are all dead.

One may thus say that some drops of antitetanic serum injected into the brain are more powerful to cure tetanus than large quantities introduced into the blood or under the skin. It does not suffice merely to administer the antitoxin ; it must be put in the right place.

Guinea-pigs rendered tetanic by means of splinters of wood may also be cured in the same way as the rabbits to which one has injected a fatal dose of toxin into the muscles.

The antitoxin introduced into the brain protects the substance of the latter although the spinal cord is already attacked by the poison, but it does not remove the lesions already accomplished ; the contractions established at the moment of intervention persist for a long time. Thus, the intracerebral injection does not save all the tetanic animals ; if poisoning of the upper parts of the central nervous axis has been effected death will not be averted. There is a time after which antitoxin can effect nothing, whatever be the fashion in which it is employed, but intracranial injection augments the period of efficacious intervention.

More than one reader will perhaps think that the brain is a delicate

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[1] Up to 10 cc. and 20 cc. in some of the experiments.

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organ, that the dilaceration caused by the needle and the compression produced by the serum injected, are capable of entailing accidents worse than those of tetanus itself. Anyone can convince himself that this is not so by repeating the experiment, which will show that in guinea-pigs and rabbits nothing is easier and less dangerous than to inject into the brain a pure liquid such as antitetanic serum. Guinea-pigs easily support eight drops in two punctures, and rabbits half a cubic centimetre. The liquid without doubt penetrates into the ventricles, and the compression determines movements of the legs in guinea-pigs and of the jaws in rabbits. But all that soon passes off, and some moments afterwards the animal eats and moves without any other impediment than that of its tetanised limb.

It is, of course, understood that we do not propose at once to inundate the brain of tetanic human beings with serum. Before that it will be necessary to multiply experiments on various species of animals, for it is possible that on horses and sheep, for example, the result might be different from that observed in guinea-pigs. An animal in which tetanus is bulbar from the outset would perhaps not be better cured by serum injected into the brain than by serum injected under the skin.[1]

Here we are considering only the cure of tetanic guinea-pigs.

IV.

CEREBRAL TETANUS AND ACTIVE IMMUNITY.

Rabbits and guinea-pigs which have passive immunity take cerebral tetanus when one injects the toxin into the brain; does the same thing happen with animals which have active immunity?

The question is particularly interesting, for many learned authorities are of opinion that the antitoxin is elaborated by the cells sensitive to the toxin, and that each system which is impressionable by the poison responds by making an antidote. One of us has supported this opinion on various occasions in his teaching. Immunity against tetanus, which is especially a nervous disease, appeared to us like an accustoming of the nerve cells to the toxin, and the production of the antitoxin a consequence of that.

It is well known what a definite form M. Ehrlich has given to this doctrine. That learned observer believes that there exists in the nerve cells a sort of central group with lateral chains, to which the toxin introduced into the body hooks itself. These lateral chains thus surcharged constitute the antitoxin, which becomes detached when it is superabundant, and penetrates into the circulation.

If the nerve cell is the source of the antitoxin of all the cells of the body it ought to be the best protected against the toxin.

Intracerebral injection furnishes us with the means of ascertaining whether this is so.

M. Vaillard has been good enough to put at our disposal two rabbits which were immunised in different degrees against tetanus, and whose serum was antitoxic.[2] After having ascertained that these animals

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[1] Up to the present time the injection of serum into the vertebral canal of rabbits has given us less favourable results than intracerebral injection.

[2] A drop of blood of the least immunised of these would neutralise ten times the dose of toxin fatal for a mouse. The precise limit of antitoxic power has not been determined.

did not experience any effect from injection under the skin of 8 cc., and even 12 cc., of tetanus toxin, which in the dose of 2 cc. was fatal to a fresh rabbit of the same weight, we introduced one-tenth of a cubic centimetre into the brain of each, and at the same time into a control rabbit.

The following day the immunised rabbits and the control rabbit showed signs of cerebral tetanus. The attacks were intense and frequent in the control subject, which died during the day. The immunised rabbits showed excitement, the characteristic mode of progression, and epileptiform attacks, repeated during the first day and less frequent afterwards. They became emaciated; the less immunised died after seventeen days, and the other after twenty days. They comported themselves like the fresh rabbits which had received only a feeble quantity of toxin.

The experiment is demonstrative; it proves that in an immunised rabbit, capable of supporting without illness large doses of toxin introduced under the skin or into the veins, and capable also of furnishing an active antitoxin, the nerve cells are still sensitive to the toxin.

. How can one admit that these are the cells which prepare the antitoxin?

It seems rather as if, during the whole course of the immunisation, they had never been in contact with the toxin. Immunity in tetanus is therefore not an accustoming of the nerve cells to the tetanus poison.

One might object that in ordinary conditions it is not the cells of the brain which take the toxin, but those of the spinal cord, that in immunised animals the latter alone are accustomed to the poison and manufacture antitoxin, and that, although it is possible to give cerebral tetanus to refractory rabbits, it is impossible to give them the true or spinal tetanus.

To reply directly to this objection it would be necessary to carry the toxin into the spinal cord, but it is difficult to do that in small animals without entailing a paralysis which prevents observation of the symptoms of tetanus. One cannot explore the spinal cord in the same way as the brain. We may remark, however, with M. Metchnikoff, that antitoxin introduced into the brain of a tetanic rabbit diffuses as far as the spinal cord and protects it; why should the antitoxin, if it exists in such excess in the spinal cord of the immunised animal that it passes into the blood, not also diffuse as far as the brain?

We intend to multiply these experiments on animals rendered refractory to tetanus and diphtheria, and to see if the same facts are reproduced in those which have attained the highest degree of immunisation.

V.

TOXINS AND NATURAL IMMUNITY.

Certain animals support considerable doses of bacterial toxins without having been gradually accustomed to them. Thus the rat experiences no bad effect from a dose of the diphtheria poison which will kill several rabbits. It has been concluded from this that the cells of the rat are naturally insensitive to the diphtheria toxin.

It is easy to ascertain whether the nerve cells of the brain are in this condition. Let us inject into the cerebral substance of a rat one-tenth of a cubic centimetre of diphtheria toxin; this dose, when introduced under the skin of another rat, does not produce even a local œdema. Nevertheless, the one which has received the toxin into the brain is soon attacked with complete paralysis. It remains paralysed for two or three days and dies.

The brain of the rat is thus sensitive to the diphtheria poison, and if this animal does not die as the result of the injection of large quantities of toxin into the subcutaneous tissue, that is because the toxin does not reach the encephalon. It is arrested in some part of the body. The natural immunity of the rat with regard to the diphtheria poison is not at all due to the resistance of its nerve cells, but to some other property of the organism.

The rabbit is supposed to be very refractory to the action of morphia; a hypodermic injection of 30 centigrammes of a salt of this alkaloid is quite well borne by an animal of 2 kilogrammes. The introduction of a single milligramme of hydrochlorate of morphia into the brain causes almost immediate effects in a rabbit of the same weight. The limbs are agitated by a trembling movement, progression is impossible; the animal remains stupefied from twenty-four to thirty hours, then it appears to improve, but it emaciates and dies in four or five days.

The nerve cells of the rabbit are thus not insensitive to morphia. When this rodent resists the injection of a large dose of the alkaloid it is because that does not reach the cerebral cells.

The facts which we have just recorded show that in acquired immunity, as in natural immunity, against certain poisons of the nervous system, the resistance is not due to an accustoming or an insensibility of the nerve cells, at least not of the nerve cells of the brain. Toxins introduced under the skin and into the blood do not attack these cells, although they have a manifest affinity for them. These poisons are without doubt retained by other cells, which play a protective rôle and probably manufacture the antitoxins. What are these cells? Perhaps the phagocytory cells which in many circumstances one sees capable of destroying the poisons contained in the bodies of bacteria. We cannot affirm that such is the case, but it seems to us that the problem of immunity against bacteria and that of immunity against the toxins will receive similar solutions.[1]

THE EFFECTS OF DEAD TUBERCLE AND GLANDERS BACILLI ON ANIMALS.

By STEWART STOCKMAN, Professor of Pathology, Dick Veterinary College, Edinburgh.[2]

THERE are two principal methods whereby the pathogenic microbes act on the animal organism. *Firstly*, by the products of their meta-

bolism in the tissues—the toxines—they poison the system. *Secondly*, they so act on the tissue cells that a lesion is produced. These two actions are in a sense related to each other, for it cannot be denied that in the majority of cases the bacterial products are largely responsible directly or indirectly for the lesion, although the purely mechanical effects of the microbes, such as embolism, cannot be altogether disregarded. The systemic effects of the toxines are of the first importance in such fatal diseases as tetanus and anthrax, but they are secondary, although important enough in the contagious diseases that tend to run a chronic course, such as glanders and tuberculosis. Of course everyone knows that acute and rapidly fatal cases of glanders and tuberculosis do frequently supervene on chronic ones, and in these we assume from the wide distribution of the young lesion, that the poisons have been administered to the system in unusually large doses.

In the majority of cases, however, these two diseases run a chronic course, and the principal inconvenience arises from the lesion. There is a great deal of work to be done yet in the study of the tissue reaction, both as regards what actually takes place and how far the bacterial products are responsible for it. With the microbes of suppuration, tubercle, glanders, actinomycosis, and some others, the initial lesion presents many points of similarity—attraction of leucocytes, degenerative changes, etc.,—but after a time they, as it were, part company, so that in many cases, though by no means in all, it is possible to say from a simple histological examination which of these microbes is responsible for a certain lesion. That is evidence of the toxines being largely answerable for the lesion, because they are special in each case.

In most cases the appearance and seat of the lesions in tubercle, actinomycosis, and glanders, when taken with the species of animal from which they have come, give us a good idea of what disease we have to deal with. The presence of a special microbe revealed by the microscope or experimental inoculation puts the diagnosis beyond doubt. It not infrequently happens, however, that tuberculous lesions appear in an organ which is much oftener the seat of actinomycosis, and *vice versa*. Take for examples, tubercle in the lower jaw of the ox, actinomycosis in the udder or lung of the cow, and the so-called clyers, which may be due to the tubercle bacillus or the actinomyces. A microscopic examination may fail to reveal the presence of any of the micro-parasites in question, and one has to fall back on experimental inoculation, which is tedious and oftimes impossible.

In the majority of cases of actinomycosis in animals we have to deal with what is believed to be an involution form of the parasite, and inoculation is useless. Moreover, if one examines lesions from an animal that has been treated with iodide of potassium, one may fail to find a single parasite, at least such has been my experience. As regards glanders, much of our present knowledge concerning some forms of the lesion is comparatively recent and dates from the introduction of mallein, which has established the glanderous nature of certain lesions hitherto misunderstood. A microscopic examination of the glanders lesion in the horse seldom reveals the presence of the *Bacillus mallei*, and the recent researches of Nocard seem to show that experimental inoculation and artificial cultivation may give negative results. Indeed,

it has been shown that mallein may fail to produce a reaction in the living animal although lesions of an apparently glanderous nature may be found at the autopsy. These cases Nocard regards as cured, and it is thus that he would explain the negative results of inoculation and artificial cultivation. The study of the tissue reaction to the dead and living bacteria, then, becomes all the more important. The lesions of glanders have been recently studied by M'Fadyean,[1] who first demonstrated the presence of giant cells in glanders nodules. Nocard[2] has studied them also from an experimental point of view. Schutz[3] has recently published the result of a very exhaustive study of the histological lesions in glanders. These papers I shall have to refer to afterwards.

Knowing that dead microbes are sometimes capable of causing a lesion, it becomes all important to know how that caused by the living differs from that produced by the dead, if it differs at all.

EXPERIMENTS WITH DEAD TUBERCLE BACILLI.

Such experiments have been undertaken before. Koch showed that dead tubercle bacilli produced an abscess in guinea-pigs when injected subcutaneously.

Prudden and Hodenpyl[4] experimented more fully on guinea-pigs and rabbits.

In their experiments they used dead tubercle bacilli from cultures on glycerine, agar, and broth. In some of their experiments they used bacilli that had been freed from their soluble products, but they found that the results were the same whether they used bacilli from agar or broth, and whether these had been washed free from their soluble products or not. They concluded that the lesions produced were due to some substance—bacterio-protein—set free by disentegration of the microbes in the tissues, or extracted in some other way. They incline more to the former view.

By subcutaneous inoculation they produced an abscess in from two to six weeks. Tubercle bacilli stainable by the ordinary methods were found in the pus.

By intraperitoneal and pleural inoculation with a milky emulsion of bacilli they produced nodules of various sizes on the serous membranes. These were made up of a central creamy looking part surrounded by fibrous tissue. The central part consisted of epithelioid cells and giant cells. Tubercle bacilli were abundant in the central part. Well marked caseation was not found.

Intravenous inoculations were made into the auricular veins of rabbits. The animals were killed and examined at intervals of from one to sixty days. A few died after the third week. In animals killed after one day the bacilli were found in the lungs, liver, and spleen, most abundantly in the first mentioned organ. The older the tubercle the fewer seemed to be the bacilli. After five days white nodules were found in the lung, some being microscopic, others quite visible to the eye. They were present up to the sixtieth day, the longest period of observation. Their structure consisted of epithelioid cells,

[1] "Journal of Comparative Pathology and Therapeutics," March 1895.
[2] Nocard, "Recueil Vet.," March 1896, November 1897.
[3] "Journal of Comparative Pathology and Therapeutics," March 1898.
[4] "New York Medical Journal," 20th June 1891.

giant cells, and leucocytes. Bacilli were found between the cells and inside giant cells. Later the nodule was denser and made up of epithelioid cells and loose connective tissue. After three weeks microscopic nodules, apparently having their origin inside the capillaries, were found in the liver. After five or six weeks visible nodules were found in the latter organ. Prudden and Hodenpyl conclude that the nodules originate in a proliferation of the vascular endothelium under the stimulus of dead and disintegrating tubercle bacilli. They say that "the dead bacilli seem to act as foreign bodies simply, curiously stimulating, it is true, but only foreign bodies after all." I think too much is made here of the foreign body question. Every foreign body does not produce this tissue reaction, although the power to do so may not belong exclusively to the tubercle bacillus. The action of the dead tubercle bacillus is in large part, at least, special to it, and it is the foreign body of this order that we are most likely to meet with in the tissues.

At the conclusion of their paper these authors offer some suggestions as to the influence of the products of the living germ on the degenerative changes in a true tubercle. They suggest, too, the possibility of the more fibrous tubercles being due to dead bacilli.

Curiously enough it was that last idea, and a wish to further test an opinion which I had formed as to the origin of tuberculous giant cells, that led me to undertake some experiments with dead bacilli. I had conceived the idea and started experimenting with dead bacilli before I knew of their paper. I have since read it very carefully, and wish here to acknowledge my indebtedness to the authors. I have performed most of my experiments on different animals, but many of the results confirm those of Prudden and Hodenpyl.

I must also mention that Straus and Gamaleia[1] have to some extent confirmed the results of the American authors by experimenting on guinea-pigs, rabbits, and dogs. They say little about lesions in the liver. They did not find giant cells in the nodules. That may be because in their experiments too few bacilli were arrested in one part, or because the centres of the nodules were not examined. Many of their animals wasted and died. When the clumps of bacilli were broken up and well distributed in the fluid the animals wasted and died all the same, but no lesions were found. Evidently several dead bacilli are necessary in one part to produce a lesion. If the number of bacilli was very small the animals wasted, then recovered and appeared quite healthy, but if a second small dose were administered they wasted and died. By using very small and ever-increasing doses of the dead bacillus they immunised the animal against it. With the products of the bacillus in artificial culture they could produce no lesion.

I would like first to say a few words about tuberculosis of the udder.

The tuberculous lesions in the udders of cows are extremely interesting, and suggest many possibilities.

If one examines a number of tuberculous udders from the cow, one generally finds distinct caseous nodules, but that is not the only form that the tuberculous lesion may assume. At the Edinburgh abattoir

[1] "Archives de Medecine Experimentale et d'Anatomie Pathologique," 1891.

during the last five years I have found a considerable number of very cirrhotic udders without any appearance of a caseous nodule in their substance. On examining these microscopically I have found tubercle bacilli and tuberculous giant cells, although the former were not very numerous. For that reason I think that the percentage of tuberculous udders has been slightly underestimated. These udders, however, will not very much increase the amount of tubercle-infected milk, because the affected quarters give little or no milk, and the cow is soon sent to the abattoir. The bacilli enter the udder by way of the blood vessels, and it is highly probable that at an earlier date there were distinct caseous tubercles in these udders now cirrhotic. Two possible explanations of the difference in these two lesions suggest themselves :—

(1.) The bacilli might have been overcome by the tissues to such an extent that they could no longer produce distinct caseation, but were still able to excite a proliferation of the tissues, and cause the formation of giant cells.

(2.) The bacilli might have arrived in the gland in an attenuated condition, and were thus no longer able to produce the distinct caseous lesion.

There is nothing wildly imaginative in these suggestions if we think on what we know of the tubercle bacillus. It is well known that the bacillus first acts by exciting a proliferation of the tissues. The new cells, instead of completing their development, however, tend to become caseous. Still, we know that the cells do sometimes complete their development and form fibrous tissue. This is especially the case in the old tubercles found in the ox and pig. They are often surrounded by a rim of fibrous tissue, which is invading the caseous centre. Moreover, the tuberculous lesions of the muscles—muscle is considered a bad medium for the growth of the tubercle bacillus— which I have described in the pig were distinctly fibrous.[1]

I may say, too, that I have once found tubercle bacilli in fibrous nodules under the mucous membrane of the fourth stomach of the ox, and have several times found them in fibrous thickenings on the sub-carpal regions of the ox. These are mainly the observations which have led me to undertake these experiments.

Experiment I.—Irish terrier bitch. I began to experiment on this animal for the purpose of trying to render her immune against tuberculosis by using the toxin and dead bacilli. I first injected her with tuberculin in doses varying from 10 cc. to 30 cc. There was no temperature reaction. I then continued with injections of the products of the tubercle bacillus in glycerine broth, from which the microbes had been separated by filtration through porcelain without previous heating. The cultures were allowed to go for from two to two-and-a-half months and longer. Beginning with 6 cc., I went to 10, 20, 40, 50, 95 cc. without disturbing the animal at all. With 95 cc. the temperature rose ·8° F., but was not above the physiological limits (102·6°). I next gave her 190 cc. into the vessels and under the skin. This toxin came from a two-and-a-half months' culture. It produced a rise in temperature of 1° F. in six-and-a-half hours. This shows the feeble systemic effect of the tubercle toxin. Thinking that the toxin prepared

[1] Stockman, "Veterinarian," March 1896.

in the ordinary way was too weak to produce much effect, for there followed no local lesion whatever—it thus differs from the powerful diphtheria toxin—I thought of trying the effects of dead bacilli. Under the skin of the chest region I injected a thick emulsion of tubercle bacilli killed by three hours' steaming. The bacilli came from a glycerine broth culture that had been kept for five months. An abscess formed and burst three days afterwards. A month later a second injection of bacilli in thick emulsion was given under the skin. The latter came from a glycerine broth culture that had been in the incubator for four months, and afterwards killed by three hours' steaming. On the third day a swelling about the size of a hen's egg formed at the seat of inoculation. The skin over this abscess was asepticised, and some pus was aspirated into a sterilised syringe by passing its needle into the cavity. Cover-glass preparations made with the pus simply swarmed with tubercle bacilli ; no other microbes were present. Glycerine agar tubes sown with the pus remained sterile. The abscess then was caused by the tubercle bacilli, which were beyond doubt dead. The abscess burst on the fifth day, and the wounds took a considerable time to heal up. The temperature rose to 105° F. on the morning after the second injection. It fell to 101·5° on the sixth day. On the afternoon of the sixth day 30 cc. of a filtered tubercle culture were injected. The temperature next morning had risen to 104·5°, and had fallen to 102° the day after. Three weeks after the second injection of bacilli I injected subcutaneously about 4 cc. of the dregs left in the preparation of tuberculin. This material was very rich in bacilli, which had been killed at about 110° C. Five days afterwards there was slight swelling and tenderness, but no distinct abscess formed. The temperature rose 1° on the day after, and fell to the normal on the fourth day.

One might draw the following conclusions from this experiment :—

(1.) That the soluble products of the tubercle bacillus produce little effect on the healthy organism, although they have a very decided action on animals whose bodies contain the tubercle bacillus, living or dead. (2.) That the dead bacilli are far more active than the soluble products, although this may be on account of their retaining a strong toxin in their bodies.

This bitch ran about and enjoyed perfect health for ten months afterwards. I was anxious to see if she would still react to the dead bacilli. On the 22nd March of this year the daily temperature ranged between 101·8° and 102·4° F. She received into her jugular vein the greater part of the bacilli from a two months' culture of the tubercle bacillus in 50 cc. of glycerine broth, which had been sterilised at 110° C.

23rd March. Temperature 105·5°. Animal rather dull.
24th March. Temperature 104°. Animal brighter.
25th March. Temperature 103·8°. Animal brighter.
26th March. Temperature 103°. Animal apparently quite well.

Up to the 3rd April the temperature fluctuated between 103·2° and 103·4° F. From the 4th to the 8th April the daily temperature at 12 o'clock was 102·4°. On the 9th April (seventeen days after), at 9.15 A.M., when the temperature was 101·6° F., 20 mm. of the dilute tuberculin were injected. My reason for starting at this hour was, of course, to get the observation over in one day.

The annexed chart will show the temperature reaction.

CHART I.

	A P R I L					
Date	9	9	9	9	10	10
Time	a.m 9 5	p.m 5	p.m 1	p.m 6	a.m 9.30 12.30	a.m 11.30

Temperature (Fahrenheit) 103° 102° 101°
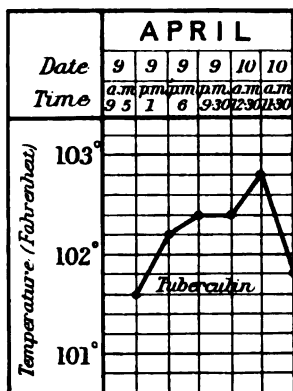
Tuberculin

The variation was 1·2°, but it did not go much beyond the physiological limits. The results of tuberculin in the dog, however, have not been satisfactory—so far at least. Cadiot records the result of the test on fifteen tuberculous dogs. The rise in temperature amounted to from ·5° to 1·7°C. In eight cases the temperature rose no more than one degree. In three cases there was no rise, but a diminution of temperature followed by death.

The animal is still alive and has always enjoyed perfect health. I am observing her to see whether she will develop a fibroid condition of the lung.

Experiment Ia.—Spaniel bitch. With a view to raising the temperature for a certain experiment of his own, Dr Noel Paton injected some dead tubercle bacilli into the external saphena vein of this animal's hind leg. He killed her twenty days afterwards, and was good enough to let me have the lungs and portions of the liver. The lungs contained many small, firm, greyish nodules, about the size of a pin's head. I have not yet examined these microscopically. The liver showed no alteration to the naked eye, but the microscope revealed small cellular collections identical with those found at the start of an ordinary tubercle caused by the living bacillus entering by way of the blood-vessels. The temperature of this animal did not rise, but I think too few bacilli were used.

Experiment II.—An aged pony was tested with tuberculin on the 17th February 1898. It did not give any reaction. The normal temperature was 99·8° F. On the 18th February two cultures of the B. Tuberculosis in 55 cc. of glycerine broth, which had been incubated for two months, were killed by submitting them to the boiling temperature for one hour. The fluid was pipetted off until the bacilli were left as a thick emulsion. As one can understand, the microbes were very numerous. At 4.30 P.M., the bacilli from the two cultures were injected into the left jugular vein of the pony with all antiseptic precautions.

The annexed chart will show the temperature reaction after the inoculation, and also the result of the tuberculin test applied three weeks afterwards.

CHART II.
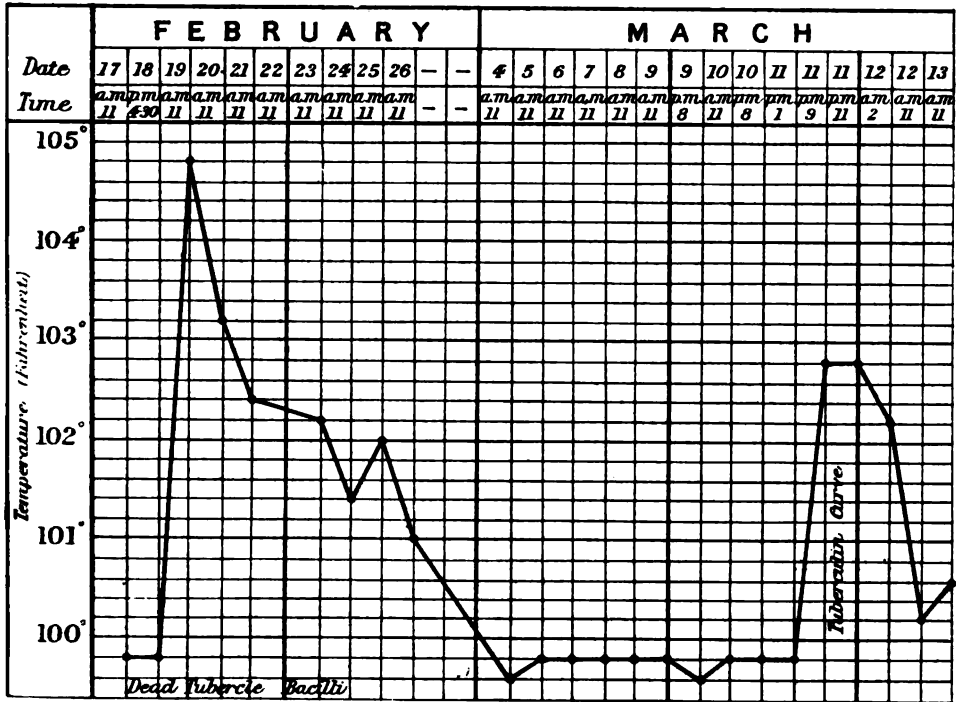
The reaction was 3° F. It will be seen that the temperature after the injection of dead bacilli fell to the normal just about the time that it should have been rising had the animal been inoculated with living bacilli. The animal was afterwards killed for the dissecting room, twenty-five days after the bacilli had been injected.

Autopsy.—The lungs were simply studded with small white nodules, varying in size from a pin point to a pea. They to some extent resembled miliary tubercles, but they differed from them in being denser and less yellow. One or two small areas about the size of a bean were very solid and of an amber colour. The latter much resembled the larger nodules that one finds in the lungs of sheep affected with parasitic pseudo-tuberculosis. There was also slight broncho-pneumonia. The jugular vein at the seat of inoculation showed a nodule in its wall. This nodule was about the size of a pea nut. It was comparatively firm, but showed one small softened centre. Cover-glass preparations, made with the softened material and stained by carbol fuchsin, showed numerous bacilli. None of the other organs showed lesions visible to the naked eye. With several nodules from the lungs guinea-pigs were inoculated into the peri-

toneal cavity.   They did not become tuberculous.   The bacilli then were dead.

Microscopic.—The lung nodules are made up chiefly of epithelioid cells and loose connective tissue, containing a good many spindle cells.   There are a few giant cells in some of the nodules.   These giant cells, however, are fewer in number and not quite so distinct as those found in natural cases of tuberculosis.   I shall afterwards state my ideas as to the significance of the tuberculous giant cell.   Numerous bacilli are present between the cells of the nodule, either as single rods or in clumps.   At some places they have assumed a somewhat circular arrangement, such as one often finds at the periphery of the giant cells in an ordinary tubercle.   The bacilli are easily stainable by the ordinary methods used for the tubercle bacillus.   There is no distinct caseation, but there are some very small homogenous patches which stain diffusely.

A few abortive giant cells and a few better formed ones are also present.   The best method of showing the giant cell is to stain with picro-carmine a section cut on the freezing microtome.   It is difficult to find them in thin sections cut in paraffin, although distinct giant cells are well enough seen in sections of ordinary tuberculous nodules cut in the latter way.   Contrary to what one finds in the ordinary tubercle, those caused by the dead bacilli are vascular.   The lumen of the smaller artery, however, is often narrowed by a periarteritis.

Vein.—The nodule in the wall of the vein has a similar appearance to those found in the lung.   The cellular centre, however, is more extensive, and there is a tendency for it to drop out of unembedded sections.

It seems to me that the giant cells are most numerous in the larger nodules, and these contain the greatest number of bacilli.

I attempted to repeat this experiment on another horse, but the animal died of embolism, in great part I think due to air bubbles in the fluid.

I have now another animal under experiment.   I will inject him with tuberculin at several intervals in order to see if he will cease to react.

Experiment III.—This experiment was undertaken to see whether a very large number of dead bacilli in one part would not produce distinct caseation or a more plentiful formation of giant cells.   For the experiment I used tubercle bacilli that had been incubated in glycerine broth for two months and then submitted to a temperature of about 110° C. for half-an-hour.   The dead culture had been kept for nine months and it was sterile.   On the 20th April a dense emulsion of these bacilli was injected directly into the lung through the chest wall, using a very fine needle.   From the 20th April to the 2nd May the temperature ranged between 100·8° and 101·3° F.   The animal was well and lively all the time.   This cat was killed by chloroform on the 23rd May, about one month from the time of inoculation.   A large nodule about the size of a pea nut was found in the lung.   The nodule was not caseous.   The pleura was quite normal.   The lung nodule was fixed in corrosive sublimate for microscopic examination.

Microscopic.—The nodule is made up of epithelioid cells and spindle cells.   There are very few giant cells, not one distinct.   The bacilli

are very numerous. There seems to be less fibrous formation than in the horse. The lung of the cat is, I think, not so well suited as that of the horse for the study of giant cell formation. Pulmonary tubercle of the cat is often in the form of broncho-pneumonia without distinct nodules, but of this I do not wish at present to make too much, as I have not had the opportunity of studying many cases.

Experiment IV.—The object of this experiment was to see whether the dead bacilli would act through the alimentary tract or not. So far as I know this has never yet been tried. The importance of such experiments is great, because once we have boiled suspected milk or flesh we consider that the danger is past. For the experiment I used a very young kitten, as they are considered easy to infect with tuberculosis by the ingestion method. On the 25th of April a kitten, six weeks old, received about 10 cc. of the emulsion used in Experiment III. This kitten must have swallowed millions of dead bacilli. She lived in perfect health until the 23rd of May, when she was killed by chloroform. All the organs appeared healthy. The mesenteric glands and portions of the ileum were fixed in corrosive sublimate for microscopic examination. They were found perfectly normal. Sections stained by carbol-fuchsin showed not a single bacillus. At present I have another cat under observation. This animal is receiving periodical doses of the dead bacilli *per os.* I also intend making some observations on the effect of the gastric juice on the living and dead microbes.

SIGNIFICANCE OF THE GIANT CELL.

It is usually accepted that the tuberculous giant cell is the result of a multiple division of the cell nucleus without a corresponding division of the cell protoplasm. By tuberculous giant cells I mean the apparently multinucleated bodies with a yellowish centre. I do not think that the multiple division theory applies to the giant cells found in animals. I cannot see how degenerating cells, for such they seem to be, could be capable of performing a function which is usually regarded as a proof of vitality, viz., reproduction.

Moreover, I have never observed mitosis in well formed tuberculous giant cells. This, however, is not an insuperable objection, as they might divide by another method.

I have seen several cells together apparently taken with degeneration in the act of nuclear division. The nuclei of these, however, were very indistinct, and their degenerate bodies had not yet completely fused. I do not think that the so-called tuberculous giant cell is really a cell, but that it is simply the commencement of caseation. I would explain its formation somewhat thus :

When the tubercle bacillus arrives in an organ it begins to multiply and stimulates the cells to proliferate ; a nodule is the result. The bacilli will be most numerous in the part where they are first arrested. Their products will there be more concentrated, and one may reasonably expect to get the most marked initial changes at that place. Now, the local action of the living tubercle bacilli on the tissues is to produce caseation as well as to cause proliferation, and the former will begin where the microbes are most numerous. The cells in the neighbourhood of the bacilli, then, will be the first to undergo caseation ; their bodies stain yellow with picro-carmine.

The nuclei resist longer, and they, along with broken up chromatin substance, can be seen for a considerable time in the degenerated patch.

It is known that the nucleins are not acted on by the gastric juice digestion, and can thus be separated from tissue cells. Possibly they are less easily acted upon by the toxins than the cell bodies, and hence their persistence. In further support of this view of the giant cell I would point out that colonies of bacilli are frequently, though not always, found around the margins of the so-called giant cells, and in the tubercles produced by dead bacilli intravenously injected in the horse the giant cells seem to be most numerous where the bacilli have been arrested in clumps. The apparent absence of bacilli from the neighbourhood of certain giant cells might be explained by supposing that the damage was due to a very small number of active ones, too few to be easily found in sections. One cannot say but what a few living bacilli may be as powerful as a large number of dead ones. Again, it might be that the tissues have overcome many of the bacilli in certain nodules and prevented the changes from going further. The dead bacilli experimentally injected certainly do in time disappear from the nodules, and we may expect those killed *in situ* to do the same. In the centre of the so-called giant cell one may find a single stray nucleus; usually one sees only chromatin substance, but even that may have disappeared. Many of the nuclei at the margin are breaking up. It is at the margin of the giant cell that they are mainly found, and they may be to the number of twelve or more. If these nuclei be carefully looked at, one often sees that they are in reality surrounded by the cell bodies in different stages of degeneration. This is best seen in the tubercles provoked by the dead microbes, possibly because the caseation is neither so rapid nor so marked as in the case of active living bacilli.

In some of the former one can see contiguous cells with caseous bodies not yet fused. Their nuclei are still present, but show up less distinctly. Some of the latter are in capillary vessels and seem to arise from the endothelial cells. As caseation extends, other cells at the periphery are included. The nuclei of the cells first struck with degeneration become disintegrated, while fresh ones are taken up at the margin. By and by the degenerating patch may increase to such an extent that the cell-like form is lost and it takes the appearance of a caseous area. Some of the cells included at the margin resemble certain of the white blood corpuscles. These may be phagocytes that have come out to surround the dead patch and have perished in an attempt to digest the bacilli.

These experiments seem to show so far that one may with impunity drink milk containing dead bacilli. That, of course, does not mean that cows with tuberculous udders should be tolerated in byres. They also suggest the possibility that some of the very chronic lesions in animals contain only dead bacilli. To decide this, of course, one would have to inoculate products obtained from a large number of these lesions in question. The production of a lesion in the abdominal organs of guinea-pigs would not in all cases be enough to prove the vitality of the bacillus, because one might possibly inject a sufficient number of dead ones to act. Artificial culture on glycerine agar, however, would decide the question.

I hope in a future paper to give the results of certain experiments on the above lines, and I think they may have a not unimportant bearing on the subject of meat inspection. The fact that I obtained a reaction with tuberculin in animals inoculated with dead bacilli says all the more for tuberculin, but I am inclined to think that the majority of animals giving a reaction have living bacilli in their bodies.

In a future paper I shall give the results of further experiments on this subject, as it is important to know about what quantity of dead bacilli must be injected before a reaction can be obtained, and how long the animals will continue to react. I am also doing some experiments with attenuated bacilli, but of these I wish to say nothing at present.

EXPERIMENTS WITH THE DEAD BACILLUS MALLEI.

My experiments with dead and attenuated glanders bacilli have not as yet been extensive. I mean at present only to describe one experiment on the horse. One of my reasons for entering into this subject at such an early stage of the investigation is, that I have found the parasitic nodules of Schütz in the lungs of two experimental ponies selected haphazard. The works of Nocard[1] and Schütz[2] are too recent to require to be quoted at any length. Nocard has produced glanders tubercles in the lungs of horses by causing them to ingest living cultures of the *Bacillus mallei*. Some of these tubercles are perfectly translucent *"les tubercules translucides,"* and Nocard asserts that in many of them the bacilli have been overcome or are in process of being overcome by the phagocytes. Some of the guinea-pigs inoculated with an emulsion made from these tubercles do not become glandered, nor can one always obtain cultures of the *Bacillus mallei* from them. Schütz, who has also done feeding experiments with glanders bacilli, found nodules due to a parasite in the lungs of many of his experimental animals and others. He hints that Nocard has mistaken these parasitic nodules for translucent glanders tubercles. What Nocard describes as " *les tubercules translucides,"* however, are dense collections of leucocytes ; one might almost compare them to small areas of white pneumonia, if I may use the expression. These, I think, are different from the parasitic nodules described by Schütz. However, it is useless to speculate on the identity or difference of certain lesions, when the matter could be decided by an actual comparison of specimens, and I think that this might form an interesting subject for discussion at a future congress.

Nobody can doubt that Schütz has frequently met with these parasitic lesions, but one is inclined to think that he must be in a badly infected area, for no other observer has so frequently noted them. Still, they are often easily passed over, unless one examines the lungs after slicing, and I must confess to having found them in the lungs of the first two horses that I examined carefully.

In view of Nocard's results, the possibility suggested itself to me, that the bacilli might be so modified by their contact with the digestive juices that they arrived in the lungs in an attenuated condition, and

[1] Loc. cit.   [2] Loc. cit.

thus fell an easier prey to the phagocytes. I do not mean to assert this, I merely put it forward as a preliminary suggestion. It might also be that fewer bacilli reach the lung when they are given by the mouth.

Experiment I.—Dead glanders bacilli. An aged pony was used for this experiment. The object was to see if the dead *Bacillus mallei* produced a lesion at all comparable with that caused by the living, and also whether the animal would react to mallein afterwards. On the 15th April at 12.15 A.M. the pony underwent a preliminary test with mallein. One might almost dispense with this precaution in Edinburgh, where glanders is a disease now rarely met with. This animal did not react. The variations of temperature will be found on the subjoined chart of the whole experiment. The mallein did not increase the agglutinating action of the serum on dead cultures of the glanders bacillus. On the 22nd April at 10 o'clock the pony received the bacilli from nine potato cultures of the *Bacillus mallei*, that had been incubated for twenty days and then killed by exposing to chloroform vapour for thirty hours.[1]

The temperature rose during the first eight hours (*see* chart), and the animal was rather seedy, but he soon recovered as the fever passed off. The agglutinating power of the serum on dead glanders bacilli was slightly increased. On the 28th April the animal was tested with mallein.[2] The temperature rose no more than 1·6° F. (*see* chart), but it remained slightly above the 99·8° F.[3] for two or three days. There was no swelling nor any systemic disturbance. The test was again applied on the 6th May at 11 P.M. with a dose of 1½ cc. of mallein. The temperature rose only 1° F. this time. There was a small local swelling about the tenth hour. It was tense and painful, but never increased beyond 2 inches in breadth, and had completely disappeared on the 10th May. The pony was killed on the 13th May.

Autopsy.—The greater part of both lungs showed very slight signs of broncho-pneumonia, and the organs were, as a whole, slightly more solid than normal.

Two lobules on the surface were the seat of hæmorrhagic infarction. The anterior lobe of the left lung was more solid than any other part, but it floated in water. On section it was of a dirty greyish and red colour. The posterior lobe of the same lung was decidedly congested, but this may have resulted from embolism. Under the pleura and in the substance of the lung were a few well-defined nodules. These were rather difficult to find in the substance of the organs, but I isolated about a dozen for microscopic examination.

They were very hard, about the size of a swan shot, and well defined from the lung tissue.[4]

When the nodules were cut across the central part resembled very dense caseous material. It was perfectly dry, and contained lime salts. At first sight I thought I had produced the translucent tubercles, but a closer examination convinced me that the nodules must have been there long before the bacilli were injected. Moreover, they were not at all like the white translucent patches described by Nocard. The

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[1] The bacilli were certainly dead, as two platinum loopful injected into the peritoneal cavity of a guinea-pig produced no effect.
[2] This mallein was kindly sent me by Professor M'Fadyean. The dose was 1 cc.
[3] This was the normal temperature of the pony.
[4] The nodules, along with portions of the lung, were fixed in 4 per cent. formol.

CHART III.

liver of this pony also showed similar hard nodules on its borders. The latter were yellower in colour than those of the lung. All were calcified. I think the parasite is the agamous form of the *Sclerostoma armatum*. In the livers of other horses I have found the same nodules, along with parasites that were beyond doubt the *Sclerostomata*. Again, in the lungs of the experimental horse that died from air embolism, I found a single nodule of the same kind after slicing the organs.

Microscopic.—The small nodules were embedded in paraffin and cut with the rocking microtome. They were found to consist of a fibrous periphery and a cellular centre. The extent of the cellular area varied in different nodules, and one may assume that the nearer the fully developed fibrous tissue is to the centre, the older is the tubercle. The most central part was invaded by lime salts. The majority of the cells were of the connective tissue type and lay in a loose fibrous matrix. A variable number of leucocytes with coarse eosine staining granules were also present. Indeed, many of the granules had been set free from the cells and could be distinctly seen outside. These coarse eosinophilous cells are a perfectly normal constituent of the horse's blood, and the shedding of the granules seems also to be a normal process. I have met with these granules often when using the haemocytometer, and my colleague, Professor Mettam[1] has lately drawn attention to their presence in the normal tissues of the horse. The granules are especially abundant in the liver nodules. Since these cells are normal constituents of the blood their presence need not be wondered at in any tissue formation. Schütz[2] says that they are absent from true glanders nodules, and nobody has yet demonstrated their presence there. It might be that this cell is not attracted by microbes. Its presence, however, can hardly be considered pathognomonic of any lesion, since it is a normal constituent of the blood.

The vessels at the periphery of the nodules are well formed, those towards the centre are embryonic. The same description may be applied to the liver nodules, except that around the periphery there is a great deal of some black pigment. All those that I have examined have had their start in a vessel of considerable size, much too large to arrest the glanders bacillus, and I do not think any pathologist would be likely to take the above formations for glanders tubercles. I have not been able to find any object that could without doubt be put down as a parasite in any of the lung nodules examined, but that is probably because I have examined only very old ones. The dead bacilli, then, had nothing to do with these formations, which I think must be identical with those described by Schütz.

The capillaries over large areas of the lung were very much distended. In some places several adjoining ones had come into contact and completely obliterated several air cells. These areas at first sight looked like commencing glanders lesions, especially as the neighbouring alveolar wall were similarly thickened. A closer examination, however, showed most of the cells to be red blood corpuscles inside capillaries, and the number of leucocytes to be about normal in proportion. No bacilli could be seen. There

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[1] "Veterinarian," May 1898.   [2] Loc. cit.

was a very small amount of exudate in many of the air cells. The latter changes were, I think, due to the bacilli injected, but nothing like a true glanders tubercle was found.

I had intended to illustrate this paper with micro-photographs, but regret that the latter arrived too late for publication. I may, however, be able to insert them in a future paper.

QUARTER-EVIL, OR BLACK-QUARTER.

By J. M'FADYEAN, Royal Veterinary College, London.

Definition.—The disease which is commonly called quarter-evil or black-quarter in England, and black-leg in Scotland, is a bacterial affection, caused by a specific micro-organism—the bacillus of quarter-evil. In France the disease is generally known by the term symptomatic anthrax (*charbon symptomatique*), originally suggested by Chabert (1782) for those cases of disease in which after a period of fever and systemic disturbance an inflammatory tumour makes its appearance in some part of the body. This author believed that quarter-evil and anthrax were merely symptomatic varieties of the same disease, and he proposed to apply the term *fièvre charbonneuse* to those instances in which the disease ran its course without the development of any tumour. Chabert is generally credited by French authors with having effected a great improvement in nomenclature when he introduced these terms, and with having thereby differentiated from charbon various other affections, such as "putrid and gangrenous fever." It is, however, difficult to understand what were the precise diseases other than anthrax and quarter-evil to which the term charbon was applied prior to the date of Chabert's monograph, and it is obvious that Chabert's definitions left it possible to confound with anthrax several other different diseases, and tended to confirm the opinion, then widely held, that anthrax and quarter-evil were not etiologically distinct.

In this country, long before the discovery of their respective organisms, anthrax and quarter-evil were recognised as things so different as to merit different names, and such confusion as exists at the present time is almost entirely ascribable to the adoption of the French nomenclature by some modern authors. The retention in France of the names proposed by Chabert may be defended on the ground that they are established by long custom, but nothing whatever can be said in favour of displacing the name quarter-evil by symptomatic anthrax. As will be shown in the course of this article, the diseases anthrax and quarter-evil have scarcely a feature in common, and in face of this fact one cannot help being struck with the curious perversity of reasoning which led the older veterinary authors to regard them as manifestations of the same morbid condition.

Discovery of the Bacillus of Quarter-evil.—Feser and Bollinger (in 1876) appear to have been the first to note the presence of rod-like motile bacteria in the lesions of quarter-evil. Both of these authors regarded these organisms as the cause of the disease, and both claimed

to have communicated the disease to cattle and sheep by inoculation. A few years later (1879 and 1880) MM. Arloing, Cornevin, and Thomas published the results of their observations and experiments, establishing the non-identity of anthrax and quarter-evil, and they described the characters of the organism encountered in the lesions of the latter disease. The last-named authors also claimed to have cultivated the organism artificially.

Morphology of the Bacillus.—The organism of quarter-evil belongs to the class of bacilli, though in some of its phases it is less distinctly and regularly rod-shaped than the causal germ of anthrax. As it is found in the tissues of the emphysematous swelling which is characteristic of the disease, it presents a certain variability in size and outline. A number of the rods—generally a minority when the examination is made some hours after death—are cylindrical, with round or slightly tapering ends, and these generally stain uniformly throughout with aqueous solution of methylene blue or other basic aniline dye. These rods are appreciably smaller, both in length and breadth, than the bacilli of anthrax, most of them being about 3-4 μ long (Plate II., Figs. 1, 2, and 3). This type of rod represents the young actively growing and multiplying organism. It is usually outnumbered by bacilli which have the form of a thin spindle, whose ends may be either symmetrical or dissimilar. The substance of the rods of this form usually stains rather faintly with aqueous methylene blue, except at one of its extremities, where there is frequently an intensely stained speck. Finally a certain number of the organisms are distinctly oval or lemon-shaped, and all gradations between these and the elongated spindle form may be found. These departures from a cylindrical form appear to represent a stage preparatory to sporulation. The organism of quarter-evil does not form leptothrix filaments in the tissues, and it is rare to find more than three rods end to end. The bacilli are actively motile when first removed from the lesions, but their movements become slow and finally cease on exposure to the atmospheric air.

The organism of quarter-evil posesses the power of sporulation, and the spores may be formed in the tissues during life, and are constantly found there after the death of the animal. In this respect the organisms of quarter-evil and anthrax are in sharp contrast.[1] The spore commonly develops at one of the poles in the case of the distinctly rod-shaped forms, but frequently its position is central. The fully developed spore is oval, with a greater disparity between its two principal diameters than in the case of the anthrax spore. It is also decidedly broader than the bacillus in which it arises, and hence when the position of the spore is terminal it gives to the rod a shape like a tennis racket (Plate II., Fig. 4).

Cultivation.—The bacillus of quarter-evil is a very strict anaerobe. Arloing, Cornevin, and Thomas claimed to have obtained cultures in bouillon made from the flesh of the fowl, and containing small quantities of glycerine and sulphate of iron. The flasks were exhausted of air, and the cultures were carried on for twelve generations. The description given by these authors, however, leaves room for grave doubts as to whether the cultures were

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[1] It has been asserted that spores are not formed until after death, but the writer has repeatedly observed sporulation in preparations made from guinea-pigs just dead from the disease.

pure, and the first successful attempts to cultivate the quarter-evil bacillus in a state of undoubted purity were made by Kitasato.[1]

The most favourable temperature for growth is from 36-38° C. No growth takes place at temperatures below 14° C., and only a sparing slow growth at 16-18°. Sporulation takes place rapidly in artificial cultures at the body temperature, and slowly at summer temperatures. The characters of the growth in different media are thus given by Kitasato.

In bouillon, in an atmosphere of hydrogen, the organism grows rapidly at the body temperature, and soon produces a general turbidity of the liquid. A little later small flocculi appear throughout the entire liquid, and these gradually settle to the bottom as a whitish precipitate, leaving the liquid clear. Gas is evolved and gives rise to a fine foam at the top of the liquid close to the wall of the flask or tube. The cultures exhale a sour odour, recalling that of rancid butter.

Cultures may be obtained in solid gelatine or agar by adding to these media while liquid 1·5-2 per cent. grape sugar, 4-5 per cent. glycerine, ·5-1 per cent. formiate of soda, or ·1-·3 per cent. of sulphindigotate of soda, to absorb all traces of oxygen. Single colonies in gelatine grow at first as irregular round balls with a slightly rough surface. The surrounding gelatine is liquefied, and radiating threads then grow out from the central mass into the liquid gelatine. In gelatine stab cultures the growth begins one or two finger-breadths below the surface, and the gelatine is slowly liquefied with evolution of gas bubbles. In agar also gas is evolved and leads to disruption of the medium. In these artificial cultures the organism presents the same forms as in the diseased tissues.

Staining.—The bacillus of quarter-evil in cover-glass preparations from the diseased tissues stains readily with any of the dyes in common use. The most characteristic picture is obtained by staining such a preparation with simple aqueous solution of methylene-blue. The bacilli take the stain instantly, and the preparation should be washed with water only, and then dried preparatory to mounting in Canada balsam. The organism is not stainable by the method of Gram. As in the case of the anthrax organisms, the spores remain unstained by the ordinary methods, but they may be coloured by exposing them to super-heated carbol-fuchsin solution in the autoclave (120° C.).

Distribution of the Bacilli within the Body.—The bacillus of quarter-evil is mainly a tissue parasite. The only place in which it is constantly found in natural cases of the disease is the emphysematous muscle tumour and its immediate neighbourhood. Here the bacillus is found in large numbers in the connective tissue of the muscle, and in the substance of the muscular fibres. The best preparations are obtained by smearing a cover-glass with a minute fragment torn from the discoloured muscle. When exudates are present in the pleural or peritoneal cavities the bacilli are present in the liquid. On the other hand the blood, the spleen, and all the internal organs, when examined immediately after death, may appear free from bacilli. Sometimes, however, the blood everywhere contains a considerable number of bacilli at the time of death. The statement by

[1] Zeitschrift für Hygiene, Bd. vi., p. 103, and Bd. viii., p. 55.

Arloing, Cornevin, and Thomas, that the bile is always rich in the bacilli is erroneous. As a rule the bile shows few or no bacilli, and even 2 cc. of it may fail to inject a guinea-pig. Quarter-evil is thus not a septicæmia, if by that term one understands a disease in which the causal organism propagates mainly in the blood and is found in large numbers there. In black-quarter, as in tetanus and malignant œdema, death usually results from the effects of toxic substances generated by the bacilli in extra-vascular positions and absorbed into the blood.

Resistance of the Virus.—A 5 per cent. solution of carbolic acid in water is fatal to spore-free cultures in three to five minutes, but an exposure of ten hours to the same solution is required to sterilise artificial cultures containing spores. Spores in artificial cultures retain their virulence after being heated for an hour at 80° C., but they are killed in five minutes at 100° C. (Kitasato). A much greater resistance to germicides is exhibited by the spores when these are contained in dried muscular tissues from a black-quarter lesion. The fresh juice or muscle enclosed in a sealed tube is completely sterilised in two hours at 80° C., and in two minutes at the boiling temperature, but if the fresh virulent muscle be quickly dried at 35° C., and subsequently mixed with water, it requires an exposure of two hours at the boiling temperature (100° C.) to completely destroy its virulence (Arloing, Cornevin, and Thomas). The dried and powdered muscle may be steamed at 100° C. for six hours without entailing the destruction of all its contained spores.

Putrefaction apparently has little effect on the virus of black-quarter, at least Arloing, Cornevin, and Thomas found the débris of muscle that had been putrefying for over six months still infective. They assert that their experiments in this direction were never successful with muscle taken thirty months after death, but guard themselves from concluding that buried carcases are always innocuous after that interval.

Habit of Life of the Bacillus.—There seems to be little room for doubt that the bacillus of quarter-evil in certain localities leads a saprophytic existence in the soil, and that, like the tetanus organism, it is only occasionally and, as it were accidentally, parasitic and pathogenic. The markedly regional distribution of the disease is easily explained on this supposition, and opposed to the view that the organism maintains its existence mainly by propagating in the animal body. At the same time, it is obvious that a single case of quarter-evil rapidly brings into existence such a number of the bacilli as are probably never formed within the same compass outside the body, and the burial of the carcase of an animal dead of the disease adds to the soil a colossal number of the germs.

Susceptibility.—The only animal in which quarter-evil appears to occur with frequency is the ox, and indeed the disease is rarely diagnosed in any other species. Nevertheless, the disease is not very rare in the sheep, and animals of that species are much more susceptible to experimental infection by way of inoculation than cattle. In the experience of the writer black-quarter has been the cause of considerable losses among sheep on farms in England and Scotland.

In sheep the susceptibility to quarter-evil persists throughout life,

but in the ox species it appears to diminish in a marked manner after two years of age. The disease is rather uncommon in cattle over that age, and in this country exceedingly rare in adult cattle. Arloing, Cornevin, and Thomas assert that this comparative immunity of adult cattle is met with only in quarter-evil districts, and that full grown animals possess no immunity in places where the disease is unknown among the young cattle. These authors endeavour to explain the alleged fact by supposing that in districts in which the disease is of frequent occurrence many animals suffer from slight attacks, not manifested by any serious symptoms, and thereby acquire immunity. The hypothesis is not very satisfying, and the evidence in support of the assertion that age brings immunity only in black-quarter districts is very slender. In this country it frequently happens that cattle bred and reared on farms free from black-quarter are transported to districts in which the disease is comparatively common, and yet few practitioners have ever seen a case in an adult animal. It is therefore probable that the escape of full-grown animals is due to an immunity which is naturally acquired with age, as is undoubtedly the case in several other bacterial diseases.

It has also been asserted that new-born calves have a considerable degree of immunity, and that this increases during the first few months of extra-uterine life, while the diet is mainly or exclusively milk, but declines when the animal takes to a herbivorous diet. These assertions are also far from being conclusively established by the available evidence, and it is quite as likely that the rarity of cases in very young calves is due to the fact that the nature of their food and the circumstances in which they are usually kept exclude some of the risks of infection. It is quite certain that among animals of other susceptible species there is no immunity during early life.

It was at one time a universally accepted opinion that quarter-evil was mainly a disease of forced or over-fed animals. This opinion has rightly been abandoned, and it is certain that the disease often attacks animals in poor or middling condition. Indeed, practitioners of experience have arrived at the conclusion that on some farms a more liberal system of feeding has reduced the prevalence of the disease.

A few alleged cases of black-quarter in the horse have been recorded, but the observations are not free from doubt. The pig appears to be immune against the disease. Among the smaller laboratory animals the guinea-pig is readily infected by subcutaneous inoculation, but the rabbit has a high degree of immunity. The fowl and the pigeon are also immune. There is abundant experience to warrant the statement that man also is immune.

Symptoms.—There are few diseases in which the clinical picture is more characteristic than in black-quarter. In animals under close observation the first symptom observed is generally lameness or stiffness of gait, and except by those well acquainted with the disease this is apt to be set down to injury. The animal is already dull and the temperature is above the normal. Appetite and rumination are soon suspended, and a swelling appears on the lame limb or some part of the body or neck. At first this swelling may not present any very special characters, but it soon becomes emphysematous and imparts a crackling sensation to the fingers when it is manipulated.

At first the swelling is manifestly painful and sensitive to pressure, but when it has become markedly emphysematous it often appears to be wholly insensitive, and may be cut into with the knife without the animal evincing any pain. Not rarely two or more swellings are formed. The animal becomes more and more dejected in appearance, the breathing accelerated, the pulse frequent. As the end approaches the patient assumes the recumbent posture, the ears and extremities become cold, the internal temperature falls, and death takes place quietly without convulsions. In rare cases the disease runs its course and ends in death without the development of any muscular or subcutaneous lesion discoverable during life. The period of observed illness in fatal cases varies from a few hours to a day, and in exceptional cases extends to two or three days.

The foregoing description applies especially to the disease as it presents itself in cattle. In the sheep the development of the emphysematous tumour is generally masked by the fleece, but the animal is sometimes observed to be lame. It lies persistently if left to itself, has a depressed anxious expression, soon becomes unable to rise, or support itself if placed on its legs, loses consciousness, and dies quietly. The duration of the disease is generally shorter than in cattle, and in many instances the animal is found dead without having been observed to be ill.

The disease is remarkably fatal, and it falls to the lot of very few people to observe recovery after the onset of the more urgent symptoms and the development of the emphysematous tumour. It is asserted, however, that in herds in which the disease frequently occurs, abortive non-fatal attacks are occasionally observed, recovery gradually taking place after the animal has for a few days displayed indefinite symptoms of illness, or even developed a slight swelling in some part of the limbs or body. It is obvious that except in the latter case the diagnosis must be conjectural, but there is nothing inherently improbable in the view that, as in anthrax, many cases of actual infection are not manifested by any pronounced symptom of illness. Systematic employment of the thermometer on all the apparently healthy animals whenever a fatal case occurs in a herd would doubtless throw light on this point.

When recovery does take place in cases in which a distinct tumour has been evolved convalescence is slow, since it involves either the gradual absorption of the necrotic tissues, or detachment of more or less skin and dead muscular tissue as a slough.

Lesions.—When the *post-mortem* examination in cases of black-quarter is delayed for a few hours after death the abdomen is found to be distended with gas, the anus may be everted, and blood-stained froth escapes from the nostrils. In the external appearance of such a carcase there is nothing to distinguish it from a case of anthrax. Rigor mortis sets in very soon after death, and putrefactive changes proceed rapidly. Over the emphysematous tumour in cattle the skin may be dry and parchment-like. In sheep the epidermis over the swollen part is generally partially shed, the wool is easily pulled out, and a sanious thin exudate exudes through the skin.

The blood in black-quarter always forms a good firm clot, this being one of the many striking differences between the disease in question and anthrax. As previously mentioned, the bacilli are frequently

absent from the blood or present in only small numbers, but both in sheep and in cattle they are sometimes present in considerable numbers, and even when the *post-mortem* examination is made within an hour after death the blood in the heart and large vessels may be frothy from the gas evolved by the bacilli. Nevertheless, even in these cases the blood clots firmly.

When the skin is removed from the neighbourhood of the emphysematous tumour the subcutaneous tissue is found to be saturated with a more or less deeply blood-tinged watery exudate. A similar exudate infiltrates the muscular tissue at the circumference of the emphysematous lesion. Within the latter more or less of the muscular and intermuscular tissues are dark in colour from congeston and actual blood extravasation, and the cut surface of the muscle has a characteristic porous appearance, due to the disassociation of its bundles by the gas evolved under the agency of the bacilli. The markedly emphysematous parts are often rather dry, and in contrast to the œdematous condition of the peripheral parts of the lesion. When the autopsy is made soon after death the escaping gas, the inflammatory œdema, and the muscular tissues are entirely free from putrefactive odour. On the other hand, the tissues of the tumour have a very characteristic sour odour, recalling that of slightly rancid butter.

The lymphatic glands in the neighbourhood of the emphysematous tumour are swollen and dark in colour from congestion.

The thoracic and abdominal viscera seldom show any notable alteration of structure, and, in contrast to anthrax, the spleen is nearly always normal in volume and consistence. Moderate quantities of blood-stained watery exudate may be present in the great serous sacs.

Microscopic examination of the muscular tissue from a black-quarter tumour reveals alterations of a comparatively simple character. The most pronounced feature in the lesion is the presence of an exudate or at many places actual blood, between the muscular bundles and in their interior. Numerous hæmorrhages have disassociated the muscular fibres and torn them across (Plate II., Figs. 5 and 6). The fibres involved in these hæmorrhages have for the most part lost their transverse striation, and the sarcous substance is more or less degenerated. Fibres not implicated in hæmorrhages may also show degenerative changes, but for the most part these are but little altered in structure, even where they bound the gas-containing spaces. The connective tissue between the bundles of muscular fibres has its lymphatic spaces distended with an exudate which is remarkably poor in leucocytes. Indeed, the almost entire absence of leucocytary reaction is one of the notable features of the lesion. As previously mentioned, the bacilli are numerously present in the tumour, and they are more abundant between the muscular fibres and in the connective tissue between the fasciculi than in the sarcous substance of the fibres. Few or none are present in the extravasated blood where that has been recently effused.

Method of Infection.—The black-quarter bacillus being in all probability a soil organism in certain districts, it would be natural to suppose that animals are usually infected by ingestion of the bacilli or their spores along with food or water. There is, however,

a great difficulty in accepting this view of infection—viz., the fact that it is difficult or impossible to infect animals experimentally by way of the alimentary canal, even when the individual selected for the experiment is very susceptible and the quantity of virus administered by the mouth enormous. At the date of the first edition of their work on black-quarter, Arloing, Cornevin, and Thomas had had nothing but failures in their attempts to infect in this way ; but in the second edition of their work [1] they claim one success. The details of the experiment, however, are in some respects remarkable, and leave considerable room for doubt as to the explanation of the result. A quantity of fresh virus was sealed up in glass tubes, which were exposed for various periods of time to a temperature of 70° C. The virus thus heated for an hour had its virulence scarcely affected, that heated for one hour and thirty-five minutes was to some degree attenuated, and with an exposure of ten minutes longer the virus was so enfeebled that it generally failed to kill. But the same virus heated for two hours at the same temperature had its virulence so exalted that it proved fatal by inoculation to guinea-pigs, and even to the rat, which is immune against the natural virus ! Feeling doubtful as to the nature of the disease induced in these circumstances, the authors mentioned administered to a six-months-old calf some pulp prepared from the muscles of a guinea-pig that had succumbed to inoculation with some of the virus heated for two hours at 70°. The calf died six days afterwards from black-quarter, and this is the alleged case of successful infection by ingestion. In view of the uniform failure of numerous other attempts, it appears possible that this was a natural case of black-quarter, and in any case the experiment does not appreciably diminish the difficulty of accepting ingestion as the common method of infection.

The only tolerably certain method of communicating the disease experimentally is subcutaneous or intra-muscular inoculation, and this suggests that a similar method of infection, acting through small wounds about the extremities, may be in operation in natural cases. To some extent opposed to this view is the fact that the lower parts of the limbs are rarely involved in the lesions, but the objection is not insuperable, for the denser connective tissue of the extremities appears to be less favourable than the loose inter-muscular tissue for the development of the bacillus, and the germs which are introduced about the feet may be carried rapidly up the limb in the lymph stream, or gain direct access to the blood-stream, and first excite a lesion when they become arrested and begin to multiply rapidly in some of the muscular masses.

It must be confessed, however, that this part of the subject is involved in uncertainty, and demands further experimentation.

In one instance which came under the notice of the writer, a few sheep were very regularly attacked with black-quarter each time that the flock was gathered together for such purposes as shearing, dipping, etc., whereas few or no cases occurred at other times. The affected sheep were usually noticed to be lame a day or two after such occasions, and on the following day they were usually found dead. In that case probably the sheep were inoculated from soil or dirt in the place where they were penned together.

[1] Pp. 119 and 140.

Diagnosis during Life.—The diagnosis of black-quarter during life is usually an easy task, for mistake is hardly possible after the development of the emphysematous swelling. The painless diffuse subcutaneous emphysema, unassociated with any imflammatory œdema, which occasionally results from the pumping of air into a wound during the movements of the part, cannot be mistaken for the black-quarter tumour, which is circumscribed, involves the deeper tissues, and is associated with an abundant inflammatory œdema at its periphery. Confusion between black-quarter and anthrax is possible when the former disease runs its course without the development of a discoverable tumour. In such a case the age of the animal and the history of the farm with reference to the occurrence of previous cases may serve as guides to a probable diagnosis. Doubt may also exist for a brief period in those rare cases of anthrax in which an inflammatory swelling is formed at some part of the body, but such swellings are easily distinguished from those of black-quarter by the fact that they never become emphysematous.

Diagnosis after Death.—Here there is seldom room for error when the examination is made while the carcase is still fresh. Cases of black-quarter without a distinct emphysematous lesion in some part of the muscular system are very rare, and when such a lesion is present it cannot, with ordinary care, be mistaken for anything else. The association of the emphysema with inflammatory œdema and hæmorrhage, the absence of putrefactive odour, and the sour rancid smell, absolutely distinguish the lesion of black-quarter.

When putrefaction has set in mistakes may be made in two different directions. Putrefactive evolution of gas may, by an inexperienced observer, be mistaken for the emphysema of black-quarter, or advanced putrefactive changes may partially mask a black-quarter lesion. The first of these errors is inexcusable, for, as already stated, the fresh black-quarter lesion has a sour but not a putrid odour. When hypostatic congestion and putrefactive changes have so altered the appearance of the tissues as to make it difficult to distinguish a black-quarter lesion, recourse may be had to inoculation experiments. A piece of the suspected muscle, or the fluid expressed from it, should be injected into the thigh of a guinea-pig, after it has been heated for a few minutes at 70° C., with the object of killing the associated putrefactive bacteria. This method sometimes immediately yields conclusive results, the guinea-pig dying in from twenty-four to forty-eight hours with a non-putrid, sour-smelling, emphysematous lesion at the seat of inoculation. Microscopic examination of a stained cover-glass preparation from the lesion will then show numerous black-quarter bacilli, which may be recognised by the characters already described. In this connection it may be observed that, while the young actively multiplying black-quarter bacilli have no morphological character by which they can be certainly distinguished from some putrefactive bacteria, the picture presented by a methylene-blue preparation from a fresh black-quarter lesion is fairly characteristic, and with a sufficient magnification it may, by anyone with a little experience, be relied upon in making a diagnosis. The guiding features of such a preparation are, the size of the bacteria, and the combination of a few regularly cylindrical bacilli, with less deeply stained spindle-shaped organisms and spore-bearing racket-shaped forms.

Microscopic examination, however, is much less serviceable in black-quarter than in anthrax.   It is hardly required when a fresh emphysematous tumour is present, and when such a tumour is not fresh the characteristics of the bacterial picture are lost.   In black-quarter microscopic examination of the blood is not reliable if putrefaction has set in, and it is of little service even when the carcase is fresh, since the bacilli are often absent from the blood or present only in small numbers.

Finally, in connection with this part of the subject it ought to be remarked that carelessly conducted *post-mortem* examination of animals dead of black-quarter leads to serious soil contamination, since at the time when the carcase is generally opened the lesions contain enormous numbers of spores, which can with difficulty be destroyed, owing to the extreme resistance that they offer to germicides.

Treatment.—It is doubtful whether any therapeutic treatment is of the slightest avail against black-quarter.   No medicinal substance introduced by the mouth can be expected to reach the seat of the disease in such a degree of concentration as will be appreciably hurtful to the bacilli, and the administration of so-called stimulants is of doubtful value in aiding the natural powers of the body to overcome the microbes.   Surgical measures are probably just as inefficacious.   The deepest scarification possible will hardly ever permit one to bring the remedy into direct contact with more than a small proportion of the organisms, and in the event of the patient surviving the attack its ultimate recovery will be rendered less probable by the putrefactive and pyogenic organisms that are almost certain to gain access to the exposed necrotic tissue.

Prevention. — MM. Arloing, Cornevin, and Thomas believed it possible to confer immunity against the disease by using as the vaccinal matter the fresh virus obtained from a black-quarter tumour. For the purposes of vaccination this material might be introduced in minimal doses into the subcutaneous tissue, or in considerable quantity into a vein or into the trachea.

The first of these methods was found to be unavailable in practice owing to the difficulty of determining for each case, in view of varying susceptibility of the individual and varying strength of the natural virus, the exact quantity necessary to avoid fatal accidents and at the same time provoke a reaction sufficient to leave the animal protected.

In the intravenous method of inoculation two parts of muscular tissue from a black-quarter lesion are triturated with one part of water, the resulting pulp is squeezed, and the liquid obtained is freed of its coarser particles by filtration through muslin, after which it is further diluted by the addition of four times its volume of water.   To vaccinate a calf 2 to 3 cc. of this liquid are injected into the jugular vein, which is exposed by dissection in order to avoid the introduction of any of the liquid into the connective tissue intervening between the skin and the wall of the vessel.   The authors state that this method of vaccinating is " certain and almost devoid of danger," and they practised it on 500 animals in 1880 and 1881, with only one death as the result of the operation.   The method, however, has since been abandoned in favour of vaccination with an attenuated virus.

Intra-tracheal injection of liquid prepared as described above was found to confer protection, but this method was never applied in practice.

In the method of vaccinating which has been most largely put into practice the material employed is muscular tissue from a black-quarter lesion which has first been dried at a low temperature, and then submitted for seven hours to a high temperature in order to diminish its virulence. The preparation of the vaccin is thus described by Arloing, Cornevin, and Thomas. The pulp obtained from a perfectly fresh black-quarter lesion is spread in a thin layer on a plate and dried at 32-35° C. The dessicated material is found to be very virulent, and it retains its strength for a very long period (one or two years). A sufficient quantity of the dried virus is triturated in a mortar with two parts of water so as to form a uniform paste, which is then spread in a thin layer on a plate, and placed in a stove previously raised to a temperature of 100-104° for the first vaccin, and 90-94° for the second. Here the material is left for seven hours, and when taken out of the stove it presents itself under the form of a brownish scale, easily detached from the surface of the vessel containing it. The material may be preserved for a long time in this condition. To prepare it for injection it is ground to a fine powder, and thoroughly mixed in a sterile mortar with water in the proportion of 1 centigramme of the dried virus to half a cubic centimetre of water. The vaccin may be introduced either under the skin of the ear or of the tail, the latter position being the one generally selected. The dose of the liquid, mixed as above, is half a cubic centimetre (= 1 centigramme of the dry powder) for a yearling calf, and a little less for younger animals. The first vaccin is injected near the extremity of the tail, on its under aspect, and the second is introduced a few inches higher up. An interval of about eight days is allowed between the two operations. The method has occasionally been modified to the extent of dispensing with the first vaccin, or operating behind the shoulder instead of at the tail or ear.

Professor Kitt of Munich proposed a modification of Arloing's method which has been practised on a considerable scale. It consists in exposing the dried and powdered virulent muscular tissue for six hours to steam at 100° C. The material thus attenuated is afterwards dried to preserve it. The dose of the dried material is 1 decigramme, and no second vaccination is practised. The method further differs from that of Arloing in that the vaccin is injected into the subcutaneous tissue near the elbow.

More recently Kitt has supplied a vaccin which contains artificially cultivated black-quarter bacilli and their spores. Here again only one operation is practised, and the vaccin is injected near the point of the elbow.

In endeavouring to estimate the economic value of these methods of protective inoculation against black-quarter the two main points for consideration are, (1) the safety of the operation, and (2) the degree of protection conferred by it.

There appears to be abundant material from which one may estimate the degree of risk attending each of the already described

methods of operating. The following table[1] supplies for this purpose most valuable statistics.

Method of Inoculation.	Number of Animals inoculated.	Accidents.					
		Deaths from inoculation.	Deaths per thousand.	Deaths from natural black-quarter after inoculation.	Deaths per cent.	Combined losses.	Per cent.
Double operation at the tail .	325,893	188	0·56	1245	0·38	1433	0·44
Total inoculations in the region of the shoulder . . .	91,066	76	0·84	365	0·40	441	0·48
Two operations at the shoulder	37,410	8	0·22	157	0·42	165	0·44
Single operation at the shoulder with Kitt's attenuated muscle powder . . .	39,084	61	1·56	187	0·48	248	0·63
Single operation with Kitt's pure culture . . .	5,643	8	1·41	81	1·43	89	1·58
Total . .	499,096	341	0·68	2035	0·40	2376	0·47

The figures quoted in the table show that in nearly half a million inoculations the average loss from the operation itself was about one animal for each 1470 inoculated, and that it was appreciably greater from the Kitt methods than from Arloing's method of double vaccination at the extremity of the tail. The figures as they stand are very reassuring, but in reality they ought to be read with others which indicate what are the occasional losses among smaller numbers of animals. Unfortunately, these are sometimes considerable, and in the case of single herds may reach to from 2 to 5 per cent.; and this even when the same sample of vaccin has been used in a number of other herds without entailing any accident whatever. In short, the black-quarter vaccin appears to be open to the same objection as the Pasteurian anthrax vaccin, and perhaps even to a greater degree, namely, that human foresight and care cannot provide a material that will have sufficient virulence to confer protection and yet not, for some individual animals, overstep the bounds of safety.[2] There are few diseases in which individual susceptibility appears to be liable to wider variation than in black-quarter, and hence arises a great difficulty in preparing a vaccin of suitable strength for all.

[1] Taken from an article contributed by Herr Strebel to the proceedings of the Sixth International Veterinary Congress, Bern, 1895.
[2] For a singular instance of serious accidents following black-quarter vaccination in Great Britain, the reader is referred to Vol. IV., page 53, of this Journal.

The second point—the degree of immunity conferred by the operation—is no less important than the first. It may be estimated in two ways, viz. (1) by experimentally testing the recently vaccinated animal with a certainly fatal dose of strong virus, and (2) by comparing the death-rate from black-quarter among a large series of vaccinated and unvaccinated animals kept in identical conditions.

The first of these methods is not so serviceable as it might at first sight appear to be. Owing to the very considerable degree of insusceptibility occasionally encountered in unvaccinated animals, such a test is fallacious unless it comprises a number of animals, including some that have not been vaccinated. The test is therefore generally inadmissible on the ground of cost.[1]

Figures given by Strebel permit one to estimate the value of protective inoculation in the second way. That author gives statistics relating to 192,866 inoculated animals and 315,168 uninoculated animals kept together on the same pastures. Of the former 840, or 0·43 per cent., died from naturally contracted black-quarter, while among the latter there were during the same period 5482 victims of the disease (1·74 per cent.). When the deaths from inoculation black-quarter were added, the losses were still three and a half times greater among the unvaccinated than among the vaccinated. If these figures are reliable—and there does not appear to be any reason to doubt them—the black-quarter inoculation must be credited with having saved the lives of a large number of animals in those countries in which it has been extensively practised, and it appears to be worthy of a more extended trial than it has yet received in this country, at least in the case of farms on which the disease frequently occurs, and where, consequently, there is strong inducement to face the risk incidental to the operation itself.

In speaking of these methods of vaccinating animals against black-quarter the dried muscle powder used in the operation is often referred to as an "attenuated" virus, but it is by no means certain that any true attenuation, such as belongs to the Pasteurian anthrax vaccin, has been impressed upon the germs present in the material. During the preliminary drying at 32–35°C. to which the virulent muscle is subjected, the bulk of the bacilli present in it probably resolve themselves into spores, and it is by no means certain that all of these retain their vitality after exposure for seven hours to temperatures of 90° or 100° C. If we assume that a considerable proportion of them are thereby killed, that would explain the apparent attenuation, that is to say, the diminished virulence of a fixed quantity of the dried muscle would be ascribable to the diminished number of living spores in it. Under this view the system of vaccination with dried muscle that has been heated to 90° or 100° C. would be equivalent to inoculation with minimal doses of the fully virulent fresh muscle juice. This view finds support in the fact that when either the first or the second vaccin is used with fatal effect, the bacilli found in the lesions give absolutely no evidence of diminished virulence. Either of the vaccins may be employed with fatal effect: (1) by using larger doses than those prescribed for vaccination, (2) by bruising the tissues at the seat of inoculation, or (3) by adding a small quantity of lactic or

[1] See report of such a test carried out by a Committee of the Midland Veterinary Medical Association, Vol. IV., page 379, of this Journal.

acetic acid to the vaccin before injecting it.   The bruising of the tissues and the addition of such agents as those mentioned doubtless act, not by increasing the inherent virulence of the spores in the vaccin, but by diminishing the resistance (phagocytosis) which leucocytes and other cells are able to offer to the multiplication of the germ within the body. .

The immunity conferred by vaccination with the heated muscle powder is supposed to last for about a year, but there is little or no reliable evidence on this point.   There can be little doubt that the protective effect is strongest a short time after the operation, and then gradually declines, but in cattle this decline is obscured by the relative immunity which is acquired naturally with age. .

Setoning, as a preventative of quarter-evil, has long been practised in this country, and many veterinary surgeons and stock-owners still retain faith in its efficacy, but it cannot be said that the evidence in its favour is at all convincing, while there is much evidence pointing the other way.   The experience on which a belief in the efficacy of setoning is founded is generally far too narrow to justify a strong conclusion either way.   The evidence offered usually is to the effect that fewer cases of black-quarter have occurred on particular farms after setoning than in previous years when the operation was not practised, or that during the same period fewer cases of the disease have occurred on farms where the young cattle had been setoned than on others where they had not been so operated upon.   It hardly needs to be pointed out, in view of the erratic distribution of the disease on different farms, and the variable number of cases occurring in different years on the same farms, that evidence of this kind is of little or no value unless it embraces very large numbers of animals. What is required, but is not obtainable, is detailed information regarding the death-rate from black-quarter among setoned and unsetoned animals grazed together or fed together in the same premises.

There is, however, a good deal of clinical evidence standing to the discredit of setoning.   In the first place, many veterinary surgeons have found it so inefficacious that they have abandoned it altogether, or practise it only at the urgent solicitation of their clients.   And with reference to this point it may be observed that a much narrower clinical experience is required to justify an unbelief in the efficacy of setoning than is necessary to justify faith in it.   A setoned animal may escape the disease although it has not acquired any protection from the operation, and hence its escape must not be immediately placed to the credit of setoning.   On the other hand, death of a setoned animal from black-quarter is positive evidence that that animal was not protected by the operation, or at least not sufficiently protected to withstand the risk of infection to which it was exposed.

In the second place, such experimental evidence as is available on the point indicates that setoning confers no protection against black-quarter.[1]

Measures of prevention ought to include, but seldom do include, the exercise of proper care in dealing with the carcases of victims of the disease.   As previously stated, such a number of black-quarter bacilli as are contained within the carcase of an animal dead of the disease are probably never formed within the same compass outside the animal

[1] See experiments by S. Stockman in this Journal, Vol. X., p. 232.

Fig. 1

Fig. 2

Fig. 3

Fig. 4

Fig. 5
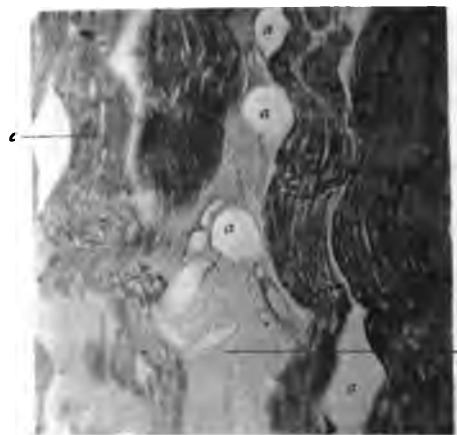
Fig. 6

body, and the burial of such a carcase, or the dissolution of an unburied carcase left where the animal died, adds an enormous number of spores to the ground. Here putrefaction cannot be relied upon to render the carcase innocuous, and it is therefore indicated to bury animals dead of black-quarter deeply in some place to which cattle and sheep will not afterwards have access.

Finally, it may be observed that improvement of the land must apparently be reckoned among the means of preventing black-quarter. Probably the most important of such improvements is draining, but ploughing and crop-growing, as opposed to leaving the land under permanent pasture, may also act beneficially. It is at any rate certain that black-quarter is now an almost unknown disease in many districts where it was at one time very prevalent, and that its decline followed the introduction of better methods of farming.

DESCRIPTION OF PLATE II.

Fig. 1. Black-quarter bacilli (non-sporulating) from a calf (× 750).

Figs. 2 and 3. Ditto (× 1000).

Fig 4. Black-quarter bacilli from a calf (× 750). The great majority of the bacilli are sporulating.

Fig. 5. Section from a black-quarter tumour in a calf (× 60); *a*, muscular fibres involved in a small hæmorrhage; *b*, extravasated blood; *c*, inflammatory exudate.

Fig. 6. Another field in the same preparation as Fig. 5, *a, a, a,* gas-containing cavities; *b*, connective tissue infiltrated with blood; *c*, muscular fibres.

EDITORIAL ARTICLES.

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REPORT OF THE ROYAL COMMISSION ON TUBERCULOSIS

SINCE the date of our last issue the Royal Commission which was appointed in July 1896 to inquire further into the subject of tuberculosis has issued its Report. At a later part of this number we have printed the series of recommendations with which the Report concludes, and we propose here to consider some of these, and the reasons for them which are embodied in the Report.

It may be well to recall the fact that this is the third occasion on which the subject of tuberculosis has been considered by a Departmental Committee or a Royal Commission. In April 1888 a Departmental Committee was appointed to inquire into pleuropneumonia and tuberculosis, and it issued its report in the latter part of the same year. In 1890 the first Royal Commission on tuberculosis was appointed " to inquire and report what is the effect, if any, of food derived from tuberculous animals on human health; and if prejudicial, what are the circumstances and conditions with regard to

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the tuberculosis in the animal which produce that effect upon man. This commission devoted nearly five years to the collection of experimental and other evidence, and issued its report in April 1895. The Royal Commission whose Report we have now to consider was appointed in July 1896, "to inquire and report what administrative procedures are available and would be desirable for controlling the danger to man through the use as food of the meat and milk of tuberculous animals, and what are the considerations which should govern the action of the responsible authorities in condemning for the purposes of food supplies, animals, carcases, or meat exhibiting any stage of tuberculosis."

The evidence taken by the Commission, relates (*a*) to the prevalence of tuberculosis among dairy stock and cattle and certain other animals destined for food in the United Kingdom ; (*b*) to the sanitary conditions under which such animals are kept ; (*c*) to the various practices governing the inspection of meat and the control of milk offered for sale, the method under which, and the extent to which, these are adopted in various districts ; and (*d*) to the alteration in the existing laws, or their administration, advocated by the representatives of various interests affected. Some evidence was also taken regarding the prevalence of tuberculosis among human beings in this country, in order to see whether any connection could be traced between that and the increased consumption of meat and milk by the population.

Statistics submitted by Dr Tatham, Superintendent of Statistics in the General Register Office, showed that between 1851 and 1895 there has been a substantial and steady diminution in the mortality attributed to tuberculosis. Unfortunately, it is impossible to accept these statistics as affording a perfectly accurate measure of the prevalence of tuberculosis during the period to which they relate, inasmuch as increasing accuracy of diagnosis, changes in nomenclature, and the different extent to which deaths have been medically certified during the period, have tended to affect the figures. But, while making allowance for this, and recognising the impossibility of obtaining really accurate and trustworthy statistical data as to the influence of the meat or milk of tuberculous animals when used for human food, the Commissoners had to note with satisfaction that death from tubercular disease in all its forms and at all ages has steadily fallen from 3483 per million in 1851-60 to 2122 during 1891-95, and that at every age-period for which statistics are available there has also been a decrease, sometimes of a very substantial character. Taking the age-period fifteen to forty-five years as the period of life when meat forms a very prominent article of diet, it is found that the diminution of death from tubercular disease has ranged from 52·8 per cent. at the age-period fifteen to twenty years, to 30·8 per cent. at the age period thirty-five to forty-five years.

These figures do not support the case of those who have been maintaining that "human tuberculosis frequently comes from the butcher's stall." Indeed, when one reflects that during the last half century there has been a great increase in the consumption of meat per head of the population in this country, and that there are grounds for supposing that tuberculosis has been increasing rather than diminishing among the animals slaughtered for food purposes, it is impossible to draw from the figures quoted any other conclusion than that the use of the flesh of tuberculous animals for human food is not a factor of any great importance in the etiology of human tuberculosis.

But Dr Tatham's statistics bring to light a very significant fact relating to the decline of tubercular disease among young children. They show that while during the age-period 0-5 years, there has been a reduction in death from tubercular disease in all forms from 5764 per million at that age in 1851-60 to 4155 in 1891-95, equal to a diminution of 27·9 per cent., the death-rate from tabes mesenterica during the same period has fallen only 3 per cent. The significance of these figures lies in the fact that the cases of tuberculosis returned under the head of tabes mesenterica are mainly those in which infection has taken place by way of the alimentary canal. The Commissioners in quoting these figures observe that the term tabes mesenterica has always comprised and still comprises a proportion of cases that are not tuberculous in their nature, but they regard it as a noteworthy fact that the rate of mortality from tabes mesenterica, which, more than any other, represents tuberculosis in infancy, has signally failed to undergo any considerable diminution during the period of sanitary progress which has been associated with such substantial diminution of death from tubercular affection at all ages in England and Wales, and that this result has coincided in point of time with a large increase in the consumption of milk.

Regarding the prevalence of tuberculosis among British and Irish agricultural stock some evidence was laid before the Commission, but hardly sufficient to enable anyone to form a very confident estimate as to the exact proportion of animals affected by the disease. The Report states that overwhelming evidence proved the greater prevalence of tuberculosis among dairy stock than among bullocks or heifers, and this is ascribed to the closer confinement of cows, to their greater average age, and to the drain on them caused by milking. The Commissioners regard the prevalence of tuberculosis among high-class pedigree stock as a serious feature of the subject, and they seek an explanation of it not in any greater predisposition to the disease, but mainly in the circumstance that pedigree cows, owing to their greater value, are retained to a greater age for breeding purposes, while their offspring are commonly subjected to more artificial treatment than less valuable stock. We do not think that there is sufficient justification

for these views, at least in so far as they imply that tuberculosis is more prevalent among high-class pedigree cows kept for breeding purposes than among ordinary milking stock. Indeed, we are inclined to think that the disease is considerably more prevalent among cows of the latter class than among those of our best pedigree stocks, but it must be admitted that the available information on this head is decidedly too meagre to enable one to institute a comparison of any great value.

The Commissioners do not share the opinion that the breed or race has much to do with liability to tubercular disease, and they note that the disease is almost unknown among cows kept chiefly in the open air, even in the case of breeds, such as those of Jersey and Finland, that evince a ready susceptibility when brought within reach of infection. With this view we cordially agree, and also with the estimate which the Commissioners formed of the practical importance of the hereditary transmission of tuberculosis. They consider that even on the estimate that three or four calves per thousand (which is the highest justified by evidence) are born tuberculous, the congenital transmission of the disease may be disregarded in practice. They consider that the main risk in breeding from tuberculous parents comes into operation after the birth of the calf, and that this should be obviated by boiling the milk before giving it to the calves. We do not believe that in this country the proportion of congenital tuberculosis among calves is as high as three or four per thousand, but even that estimate would fail to invest the congenital method of transmission with any practical importance when one has to consider methods for combating the disease. It must be remembered that in all probability a considerable proportion of the calves that are born tuberculous succumb to the disease soon after birth. Furthermore, the owner of a valuable cow that has reacted to tuberculin but gives no other evidence of being affected with the disease may calculate that if he breeds from her the chances are three hundred to one against her calf being born tuberculous, and when the calf is born he can ascertain by the tuberculin test whether it is tuberculous or not.

It need hardly be said that all the evidence laid before the Commission was not to this effect. Some of the witnesses very strongly deprecated breeding from tuberculous animals, not on the ground of *post-partum* risk of infection, but because they attached a great importance to the intra-uterine transmission of the disease itself, or to the hereditary transmission of a special susceptibility to infection. But no stronger justification for these views was forthcoming than that they were widely held by breeders, and apparently those who put them forward entirely overlooked the fact that in the meantime there is no alternative to breeding from tuberculous cows save the sacrifice of from 30 to 40 per cent. of the breeding stock in this country.

On the question of the danger which arises to human beings from the fact that tuberculosis is a common disease among cattle slaughtered for the food of man, the views enunciated by the most recent Royal Commission are to a large extent reassuring, both to the general public and to farmers and butchers. The Report endorses the finding of the first Royal Commission, that "any person who takes tuberculous matter into the body as food incurs risk of acquiring tubercular disease," but it records the opinion that the risk to the human subject of acquiring tuberculosis through meat has been greatly over-estimated. The members of the second Commission could find no indication of this danger in the mortality returns already quoted, and the only evidence pointing to such a danger with which they were acquainted—namely the results of certain artificially contrived infections of meat made at the instance of the previous Commission—was the outcome of deliberately contrived laboratory experiments, admittedly carried out under methods involving a risk greater than any that would arise in ordinary trade procedures. The Commissioners, while thus refusing to adopt the extreme views regarding the danger associated with the sale of the flesh of tuberculous animals which have at times been put forward both in this country and abroad, do not deny that some danger of human beings becoming infected through tuberculous meat actually exists. The danger, they think, has been exaggerated or over-estimated, with the result that in some places a good deal of meat has been needlessly condemned. They are not prepared to recommend that indiscriminate traffic in tuberculous meat should be permitted, or that inspection should be more lax than it is at present. On the contrary, they think that inspection of meat as to its fitness for human food should be general, and that some general principles for the guidance of meat inspectors in dealing with the carcases of tuberculous animals should be authoritatively laid down. "Chaos is the only word to express the absence of system in the inspection and seizure of tuberculous meat." The Commissioners found that the stringency of the principles governing the condemnation of carcases in different places varied in the most extraordinary way, apparently according to the whim of the medical officer or veterinary inspector. The absurdity of this haphazard method of conducting meat inspection has on several occasions been pointed out in these columns, and we heartily welcome the prospect of a more rational system.

The Report of the Royal Commission says that such a stage of experience and knowledge has been attained as to the nature of tuberculosis and the effect of tuberculous meat upon the human consumer, as to enable a uniform standard to be prescribed for the guidance of meat inspectors, and among its recommendations there is embodied a sketch of the principles which, in the opinion of the Commissioners, should be observed in judging the carcases of tuber

three thought that compensation ought to be awarded. The grounds on which the opinion adverse to compensation was founded were— (1) That the risk of seizure and condemnation on account of tuberculosis is one that is fully recognised in the trade, and necessarily affects the price paid for the living animal ; (2) that even in the past the loss thrown on butchers has not been so great as has often been represented ; (3) that the loss will be smaller in the future if effect be given to the recommendations made by the Commission with regard to the extent of disease justifying seizure ; and (4) that evidence laid before the Commission had convinced them that the losses incurred by seizure can best be met by the system of mutual insurance.

The Commissioners who are in favour of compensation for condemned carcases admit that it cannot be claimed on the ground of any analogy with that which is granted in the case of such diseases as pleuro-pneumonia and swine fever, because in the seizure of tuberculous carcases there has neither been notification nor compulsory slaughter. Nevertheless, they think that a claim for exceptional dealing here arises from the fact that the butcher is absolutely irresponsible for the condition that entails condemnation, and because it does not appear to be possible to bring the loss home to the farmer or grazier. This, of course, assumes that the danger of an apparently healthy animal turning out to be extensively tuberculous is not taken into consideration by the butcher, for, if it is, he obviously incurs no loss. And we find it difficult to see why, where condemnation of carcases is a frequent occurrence, butchers should fly in the face of sound commercial principles, and offer more for the live animal than past experience justifies. At any rate it is clear that if they do they have no well-founded claim to compensation. It is an entire misrepresentation of the circumstances when the butcher demands compensation because his "good carcase" has been condemned in the public interest. The accurate statement of the case is, that he has come into the possession of an article which he supposed to be good, but which is valueless for the purpose to which he intended to put it, or which cannot be put to that purpose without doing injury to others. To admit the claim for compensation in these circumstances would be to sanction quite a new principle, and one that would probably prove very expensive, for under it not only tuberculosis but almost every other morbid condition for which the flesh of slaughtered animals has to be condemned would entitle the butcher to reimbursement.

We entirely agree with the opinion that if there is any loss it ought to fall on the breeder or feeder who owned the animal at the time when it contracted the disease, and it is a pity that, owing to the complicated nature of the cattle trade, and the repeated sales that often take place between the time when the animal leaves the

farm and arrives in the slaughter-house, it is well-nigh impossible to place the penalty on the right shoulders. It is no longer possible to regard the prevalence of tuberculosis in a herd as a pure misfortune, in the sense that human foresight is powerless to prevent the disease or stamp it out ; and to grant compensation for the condemnation of tuberculous carcases seized in slaughter-houses would inevitably tend to make farmers negligent in the exercise of measures of prevention.

The chorus of disapproval with which the terms of the Report of the Royal Commission have been met by some representatives of the farmer's and butcher's interests would almost justify the belief that the members of these callings consider the granting of compensation for the seizure of carcases the only thing necessary to settle the tuberculosis question. For the reasons above stated we consider that it would probably prolong it, but in any case it is ridiculous to maintain that the whole of the recommendations of the Royal Commission are likely to be rendered nugatory by the refusal to grant compensation. There is no doubt that the outcry raised in favour of compensation is out of all proportion to the value of the carcases condemned on account of tuberculosis. Notwithstanding numerous requests to be supplied with evidence as to actual cases of substantial loss incurred in this way, hardly any of the witnesses who came before the Commission could tell of more than some two or three such seizures during long periods involving sales of hundreds and even thousands of carcases. A fair measure of these losses is found in the fact that even in places where the system of inspection and condemnation is exceptionally and unnecessarily severe a premium of 3d. per head for bullocks and 6d. per head for cows proved quite sufficient to cover the losses through condemnation of tuberculous carcases.

The compensation which we have been discussing does not refer to that claimed for the condemnation of live animals showing symptoms of tuberculosis. That is an entirely different question, which we propose to consider on a future occasion along with other matters dealt with in the Report of the Royal Commission.

THE MICROBE OF PLEURO-PNEUMONIA.

FOR many years the bacteriological methods which have sufficed to reveal the germs of a considerable number of the contagious diseases have been applied to the lesions of pleuro-pneumonia, but with uniformly unsuccessful results. No one at the present day doubts that in the case of every contagious and infectious disease a living germ is at work, and is equally responsible for the progress of the disease in the sick animal and for the spread of the disease from one animal

to another. It has long been known that the inflammatory liquid which saturates the diseased portion of lung in pleuro-pneumonia is capable of setting up a precisely similar inflammatory process in the subcutaneous connective tissue when it is injected into an experimental animal; and for this purpose even a small quantity of liquid from a diseased lung suffices. This liquid, it was felt, must therefore contain the germs of pleuro-pneumonia, probably in considerable numbers, and yet they could neither be directly detected in it with the microscope nor cultivated from it in any of the artificial substances which serve as a quite appropriate soil for the growth of such germs as those of anthrax, glanders, or tuberculosis. At last, however, MM. Nocard and Roux[1] appear to have solved the problem, and their success is mainly due to the adoption of a method of cultivation which very closely assimilates the artificial to the natural.

When natural products, such as pus, blood, or inflammatory liquid, containing a few disease germs are introduced into the tissues of a susceptible animal, these germs begin to multiply at the place where they were introduced; but this method of experimentation affords little or no assistance in the search for the disease germ if that was not detectable in the material used for inoculation. As the disease develops where the material used for inoculation was introduced, we have the most confident conviction that the germs are multiplying there, but when we come to explore the inflamed tissues they escape our observation, because of their small size and of their being concealed by tissue elements with which they are mixed up. About two years ago MM. Metchnikoff, Roux, and Salimbeni, in carrying out some researches on cholera, had recourse to a method which enabled them to cultivate the organisms in the body of a living animal, in a liquid protected from invasion by those wandering cells which in ordinary methods of inoculation generally flock to the place where the germs are growing. These wandering cells have the double disadvantage of interfering with the multiplication of the germs and of concealing them from view when attempts are made to discover them with the microscope. The authors mentioned employed little capsules of collodion filled with a sterile nutrient clear liquid (the ordinary *bouillon* of the bacteriological laboratory). After adding to the liquid in the capsule a little of the material containing the germs (to serve as seed), the aperture was carefully closed, and the capsule thus sealed was introduced into the abdominal cavity of a living animal, and left there for a period of days, months, or weeks.

Across the wall of such a capsule the imprisoned microbes cannot pass in the outward direction, nor can the leucocytes, or other animal cells which are attracted by the microbes, pass through it in the opposite direction. Nevertheless, the collodion film is not a barrier to the diffusion of liquids, and hence it happens that by interchanges

[1] " Annales de l'Institut Pasteur," Vol. XII., p. 240.

through the wall of the capsule its contained liquid gradually becomes assimilated in composition to the natural lymph, and the germs thus find themselves, in respect of nutriment, just as favourably circumstanced as if they were growing freely in the tissues. Moreover, those excrementitious products which the bacteria themselves manufacture, and which tend to hamper or arrest their growth, also diffuse out of the collodion capsule, and never reach that degree of concentration which would be hurtful to them. In short, it may be said that the same method if applied to pisciculture would consist in breeding fish in their natural habitat, in a net with meshes too small to allow them to pass out, and yet large enough to permit of their food materials passing in.

MM. Nocard and Roux introduced such collodion capsules, previously inoculated with a trace of liquid from a pleuro-pneumonia lung, into the abdominal cavity of rabbits, and left them there for fifteen to twenty days. When they were removed it was found that their contained liquid, originally quite clear and transparent, was now slightly albuminous and opalescent. Under the microscope it showed no cells, but with a magnification of about 2000 diameters an infinite number of small refractile moving points could be made out. A similar collodion capsule filled with the same nutrient liquid, but not inoculated with the virus from a pleuro-pneumonia lung, was found to be quite transparent and devoid of any of these moving particles after a sojourn in the abdominal cavity. That the moving particles were really living things was proved by ascertaining that a trace of liquid containing them from the first capsule could be used as the seed material for a second, which after a sojourn in the abdominal cavity again showed an infinite number of moving particles in its liquid. If, however, the liquid containing these particles was heated so as to kill them, then when used as a seed material to another capsule the latter remained quite clear and transparent after incubation in the abdominal cavity of a rabbit.

The proof that the germ of pleuro-pneumonia multiplies in the liquid of these collodion capsules, and that the minute refractile motile particles are the actual germs themselves, rests on the results of certain inoculation experiments on cows. The liquid from a pleuro-pneumonia lung when injected under the skin of the body of an animal of the ox species is capable of setting up a lesion which is always serious and often fatal. MM. Nocard and Roux found that the contents of the incubated capsules had the same effects. Five cows were thus inoculated, with the result that more or less formidable swellings formed at the seat of inoculation in all of them, and one of them succumbed. Three of the survivors were afterwards inoculated with virus from a pleuro-pneumonia lung, and experienced no bad effect, thus showing that the first operation had left them protected against pleuro-pneumonia, and furnishing another link in the chain of proof that the

refractile moving particles in the incubated capsules were the actual germs of the disease. The authors are unable to give any information regarding the form of the germs, as even the highest powers of the microscope scarcely do more than make them visible. It is interesting, however, to know that, besides cultivating the germ in the manner already described, they also succeeded in growing it outside the body in test-tubes, the artificial medium employed being a solution of peptone to which a small proportion of serum of the blood of a cow or a rabbit had been added.

A great interest attaches to these researches because of the hope which they hold out, that the application of similar methods will make us acquainted with the hitherto unknown germs of other equally important diseases, and in this connection it may be noted as a singular fact that the germs of all the most intensely infectious diseases of man and animals, such as small-pox, measles, cattle-plague, and foot-and-mouth disease, are still unknown.

Review.

Annual Reports of Proceedings under the Diseases of Animals' Acts, the Markets and Fairs (Weighing of Cattle) Acts, etc., for the Year 1897.

IN addition to the usual statistical tables, maps, etc., relating to the incidence of the scheduled contagious diseases of the domesticated animals during the year 1897, this annual publication includes reports by the Chief Veterinary Officer and the Assistant Secretary in the Animals' Division of the Board of Agriculture. The bulk of the Report by the Chief Veterinary Officer is devoted to an account of the history and morbid anatomy of pleuro-pneumonia. This is illustrated by a series of coloured plates, which very faithfully reproduce the naked-eye appearance of the lesions commonly met with in a pleuro-pneumonia lung, and add greatly to the interest and value of the Report. The account of the pathology and morbid anatomy of the disease is based mainly on the paper contributed to the Journal of the Royal Agriculture Society a number of years ago by Dr Yeo, who attempted to refer some of the most characteristic features of the lesion to a mechanical stagnation of lymph in the lymphatic vessels of the lung, following upon a chronic ulcerative bronchitis. We think that this is an entirely erroneous account of the pathology of pleuro-pneumonia.

We observe in the section devoted to the etiology of pleuro-pneumonia a statement that has often been made before, but which is nevertheless open to very serious question. It is that "it appears to be very probable that the only means by which pleuro-pneumonia is transmitted is the inhalation by the healthy of the breath of a living diseased animal." Now, it is true that cohabitation of diseased and healthy animals in the same premises appears to be the only way in which the disease is naturally spread, but what is the justification for the statement that inhalation of the breath of a diseased animal is necessary for infection? Experience certainly does not show that among a lot of stalled animals the disease always first attacks those on either

side of the diseased one, and when a diseased animal is stalled in the midst of healthy ones it is quite impossible to predict which of these will be first attacked. It is almost certain that it is not the breath *per se*, but solid or liquid particles expelled from the air passages, that contain the germs of the disease, and probably the method of infection is just the same as in some other diseases, viz., by the inhalation of such germ-containing particles suspended in the air.

In the Chief Veterinary Officer's observations on glanders we find a paragraph to the effect that horse-owners can hardly be expected to consent to the slaughter of animals that have reacted to mallein but present no external evidence of disease, unless they receive their full value. This is equivalent to an expression of opinion on the part of the Chief Veterinary Officer of the Board that the condemnation of apparently healthy horses, on the sole ground that they have reacted to mallein, requires the consent of the owner. This is important, for in London some over-zealous veterinary inspectors to the Local Authority a short time ago displayed an inclination to treat every horse that had reacted to mallein precisely as they would one clinically glandered, and some horse-owners have since then hesitated to use mallein. The paragraph referred to will probably reassure them on the point.

Many other interesting points relating to the contagious diseases are discussed in the reports of the Chief Veterinary Officer and the Assistant-Secretary, but want of space forbids our referring to them here.

CLINICAL ARTICLES.

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PSOROSPERMOSIS OF THE INTESTINES IN CATTLE.

By GEORGE GAIR, M.R.C.V.S., Conon Bridge, N.B.

THE following facts relating to an obscure disease which came under my notice some time ago may be of interest to the profession. I do not remember of its being recorded in our journals, or treated in our text-books.

The outbreak of this disease occurred on a farm composed of a sandy loam, situated on the seaboard, sloping south-east, and hitherto considered perfectly healthy for stock. The present tenant came into possession early in summer, and all went well with his herd till the beginning of July following, when he observed one of his cows unwell.

This cow, a fine five-year-old polled Angus, was bred by the owner, and in excellent condition, and at this date had been just one month on the farm. The symptoms which most attracted the attention of the owner were, back highly arched, hair standing, great stiffness in walking, feet slightly swollen from knee downwards, and from hock downwards in hind feet, the swelling terminating very abruptly immediately above knee and hock, and the animal appearing greatly pained when made to walk. One of the most prominent symptoms was the daily passage of blood-stained fæces, although the blood was not present in large quantities. Suppuration of the udder took place

during the attack, which lasted about one month, when recovery took place.

The next case was that of a yearling, which became affected about the beginning of July also, and showed exactly the same symptoms as the previous case. The attack, however, was a milder one, recovery taking place in about a fortnight.

The next two cows affected with the disease were both pregnant with first calf. They calved during their illness, and shortly after birth both calves died. These two cows displayed the same symptoms as the foregoing cases, with this distinction, that their udders were more severely affected, and discharged a great deal of bloody pus. Both animals recovered after a month's illness.

On the first of August another cow was attacked more suddenly and with greater severity than any of the previous cases, a larger quantity of blood being passed in the fæces, especially during the last three days. This cow died in about twelve days after the first appearance of illness.

Up till the end of autumn four other cases occurred, all of which ended fatally. I am informed by the owner that the symptoms were not so well marked, especially the swellings in the limbs, nor was the blood-stained fæces so prominent a feature as in the previous cases, until perhaps a day or two previous to death. Possibly this may be accounted for by the fact of the disease running a very acute and rapid course.

No more cases occurred until the first of April following, when I was consulted for the first time in regard to this serious outbreak of disease. On arrival at the farm I found a two-year-old bullock suffering evidently from the same disease which had affected the cattle during the previous summer and autumn. The animal was lying down, and apparently had been several days affected. It was with considerable difficulty made to rise. The arching of the back and the staring of the coat were well marked, as in the cases already mentioned, the prostration was excessive, and there was evidently great pain on being made to move. The lameness was particularly well marked in this case, the hind leg becoming affected first. In about a week one of the fore legs became similarly affected, and marked swellings in both limbs followed quickly thereafter, until they obtained fully twice the natural size. A peculiarity of the swellings was the abrupt termination immediately above the knee and hock. In this case blood began to show in the fæces between two and three days previous to death, and was passed in large quantities the day before the animal died. The duration of the attack was about a fortnight.

All the animals had a difficulty in rising or lying down, and each of them exhibited the swellings more or less on the legs, but nowhere else on the body. They would on an average be affected about eight days previous to blood being noticed in the fæces.

I had no opportunity of making a *post-mortem* examination of any of the animals which died, with the exception of the last one affected, the internal organs of which were removed immediately after death and carefully laid aside until they were seen by me about five hours afterwards. The bowels seemed to be the principal seat of the disease ; they were extensively inflamed, and partly filled with bloody fæces. A large clot of very bright red blood was taken

from the chest, where it lay between the lung and the chest-wall, to either of which it seemed to have little or no attachment.

The disease evidently being of very rare occurrence, I forwarded a portion of the bowels to Principal M'Fadyean, who, after microscopic examination, attributed the disease to a minute animal parasite, the coccidium oviforme.[1]

As a preventative sulphate of iron was given in food to the remaining animals, and no further outbreak occurred.

HUNTING ACCIDENT—FRACTURED SCAPULA.

By J. G. PARR, F.R.C.V.S., Leicester.

ACCIDENTS to horses in the hunting field are not uncommon, but I trust the following account, which came under my notice this season, may be interesting to readers of the Journal.

It illustrates how very difficult it sometimes is to give a correct opinion on the spot, and it shows what a serious condition can be maintained for a long time before a true diagnosis can with safety be formed.

The subject of the present note was a very valuable chestnut mare, belonging to a hunting gentleman from the Pytchley Hunt, and having a day with the Quorn Hounds on 7th March 1898.

During a gallop I noticed the mare at a standstill in the centre of a field, and on going up to her I found that she was quite unable to move, and seemed in great pain. The near shoulder was swollen all over the surface of the scapula, right down to the elbow, so that it was almost impossible to hear any crepitus, or to tell if the shoulder was dislocated. The latter condition seemed to me unlikely from the fact that I could move the leg fairly well, although on flexing it past a certain point the mare evinced pain. I had the mare taken into a stable, which luckily was only one field away, and although it required both time and patience I managed to get her to hobble along. She was left in charge of myself and Mr Tinkler, V.S. of Syston, who kindly assisted me throughout the case. A man was set to foment, and the fomentations were continued for two days and two nights. The mare was put in slings, and ordinary treatment adopted.

At the end of a week the mare was feeding well, the swelling had subsided, and on careful examination I came to the conclusion that there was a fracture at the neck of the scapula.

It then became a question as to whether the animal was worth keeping, but as she was a very valuable mare the owner thought it

[1] The piece of large intestine forwarded by Mr Gair showed patches of intense inflammation without ulceration. An unsuccessful search was made for small nematode worms, which, it was suspected, might have been the cause of the inflammation, and during this search coccidia were detected in the scrapings from the bowel. Further examination showed that these were present in great numbers in the inflamed areas of mucous membrane. The case appears to be the first of its kind recorded in this country. A translation of an interesting article by Herr Zschokke on dysentery of cattle, caused by a coccidium, appeared in this Journal, Vol. V., p. 101. Some of the symptoms noted in Mr Gair's cases were not observed in the Swiss disease, notably the painful swelling of the legs. It is difficult to explain the connection of that condition with the coccidiosis of the intestine—J. M'F.

would be worth trying to get her round, if only for breeding purposes.

The mare seemed well in health, was feeding, and showed little if any sign of pain. I saw her every few days, and thought the case progressing favourably. On 2nd April I was surprised to hear that she had died in the night, and been buried at once.

Feeling a great interest in the case, I requested to have the bones sent to me, and this was done, but the decomposition was so great that it was a very unpleasant task, and was not done as carefully as it might have been, part of the bone being left on the carcase. Examination showed that the scapula had been fractured a short distance above its articular angle, the lower end of the bone being resolved into three pieces, one of which comprised the neck and glenoid cavity. Judging from the appearance of the bone after removal from the body, one would have thought that diagnosis ought to have been easy.

Another interesting point is as to the cause of the injury. The mare jumped all her fences without a mistake or the slightest suspicion of hurting herself. No fence had been taken for more than two fields, when the rider in galloping across a grass field heard something, as he describes it, " go crack," and the mare then became dead lame.

REPORT ON AN OUTBREAK OF RINDERPEST.

By P. SCOTT FALSHAW, M.R.C.V.S., Singapore.

ON the 7th April 1897, I was asked by the proprietor of Siklap Estate to examine some bullocks that were ill there. On arriving at the estate, which is a cocoanut plantation, I found that two bullocks had died the day before from " diarrhœa," and that four were very ill. These four cattle were feverish, weak, and could hardly stand ; they were purging, and after violent straining passing slimy watery fæces containing blood.

There were also fifteen other bullocks on the estate, but the four sick bullocks had been separated from these by the manager on their showing symptoms of disease, and isolated about 600 yards away in another shed.

On the 9th I shot one of the diseased bullocks, which was nearly dying, removed the gall bladder, emptying the bile into a sterilised vessel, and injected hypodermically the remaining fifteen bullocks with it, with the exception of one (No. 6) which had a very high temperature and was isolated with the four sick animals.

Of the latter animals, one was shot on the 9th, one died on the 10th, one on the 11th, and the remaining one on the 12th.

No 6 showed all the symptoms of rinderpest—high temperature, rough coat, drooping head, not eating, purging, fæces containing blood, eruption on the palate and gums, and discharge from eyes and nose. He died on the 13th at 2 A.M.

[TABLE.

TEMPERATURE CHART.

No.	Breed.	Sex.	Condition.	Temp.	Bile.	8.30 a.m.	3 p.m.		11 a.m.	Noon.		Noon.
						April 9th 1897.			April 10th. 11th. 12th. 13th. 14th. 15th.			
1	Siamese	Bullock	Good	103·4	10 cc.	104	103·8		102	102		101·8
2	,,	,,	,,	104	9 cc.	103	104		102·2	102·8		102·4
3	Indian	,,	Poor and old	103·6	8 cc.	102	103		101·6	101·4		101·8
4	Siamese	,,	Fairly good	102·5	10 cc.	102	102·8	Temperature not taken, thermometer broken, all well.	102	102	Temp. not taken, all doing well.	103·6
5	,,	,,	Old, fairly good	102·4	10 cc.	104	—		102	103·2		102
6	,,	,,	Off feed—diarrhœa	106·4	isolated.		very ill		Died 2 a.m. 13			—
7	,,	,,	Fairly good	103·4	10 cc.	103	103·4		103	103		103·2
8	,,	,,	,,	105	not injected.		102·6		101·8	101·6		101·8
9	,,	,,	,,	102·6	10 cc.	102·4	—		101·8	101·4		102·4
10	,,	,,	,,	103	10 cc.	103·4	103·6		102·6	102·8		102·6
11	,,	,,	,,	102	10 cc.	102·2	—		101·4	102		102·8
12	,,	,,	Poor	103·4	10 cc.	102·8	103·4		103	102·6		103
13	,,	,,	,,	104·6	not injected	102	103·8		103	101·8		101·6
14	,,	Bull	,,	103·4	not injected	102	103		102	102		102·6
15	,,	Bullock	Good	104		102	—		101·6	101·8		102·2

The seat of inoculation was behind the shoulder. On the morning of the 10th there was in every case a large, diffuse, soft, and painful swelling at the seat of inoculation, as much as 4 inches in diameter, and causing lameness.

On the 11th the swellings were very hard and painful.

On the 12th the swellings in most cases were more circumscribed and not so painful; animals still lame.

On the 13th swellings much smaller, and not so painful; all eating and looking well.

From the 14th to the 19th the swellings gradually decreased, until by the 20th they were hardly to be detected.

All the animals were kept indoors during the whole time, and were all eating well. The inoculation did not in any way interfere with their general health.

On the 24th April I removed Nos. 4, 12, 13, and 14 to the Quarantine Station, and on the morning of the 25th I injected hypodermically 1½ cc. of blood freshly drawn from the jugular of a bullock affected with rinderpest.

	25th April, 5 p.m.		26th Apr. 9 a.m.	26th Apr. 5 p.m.	27th Apr. 7 a.m.	27th Apr. 5 p.m.
No. 4	100·4	102	102	102	100·8	104
,, 12	100·8	102	103·4	103·6	100·6	103·6
,, 13	98·8	100·6	99·4	101·6	98·4	102·4
,, 14	101	100·8	101	101·6	98·8	102·4

The bullock from which the blood was drawn was purging badly, and passing blood, with discharge from eyes and nose, and an eruption on the palate. Temperature 102°.

On the 25th, 5 P.M., no swelling at seat of inoculation.

26th, all eating well except No. 14, which is off feed.

The bullock from which the blood was drawn is looking better and eating.

M

27th, all eating well. The bullock from which the blood was drawn is eating well, and his dung is solid.

These four bullocks were kept in a shed where several other cattle were affected with rinderpest, and some dying daily. Three other cattle that came with the bullock which furnished the blood used to inoculate the above four animals all died, it being the only one that recovered.

No 13 had a very large abscess at the root of the tail, involving the sacral region, and it died on the 29th from the exhaustion caused by this abscess.

Nos. 4, 12, and 14 were discharged from the Quarantine Station on the 10th May, having remained healthy all the time, and I believe they are still alive.

FRACTURED RIBS.

By FRANK G. ASHLEY, M.R.C.V.S., Bath.

CASE NO. 1.

ATTENDANCE was requested to a favourite thoroughbred horse about twenty years old, which had slipped down broadside when at walking exercise on the rein. On my arrival an hour subsequently I learnt that he had fallen on the near side, and that he could not be persuaded to try to get up although he was turned over; but after having his head bathed with cold water for some considerable time he got up of his own free will, and walked home without exhibiting any ill effects from the fall. On examination I noticed a little flatness of the near hind quarter, and on running the hand over the chest wall a slight swelling was detected over the middle of the eighth and ninth ribs on the same side, but it was not thought of any serious importance as it was not painful on pressure. On his being made to move it was noticed that the near hind limb was carried stiffly, but no fracture of the anterior iliac spine could be detected. I directed that he should be kept on the rack chain and be given a laxative diet of bran and linseed mashes, with grass at intervals, hot fomentations and soothing liniment to be applied to the near hind quarter, his bed to be cut short so as not to interfere with the free movement of the near hind limb. It was not thought advisable to put him in slings, as he was of a very nervous disposition.

Second Day.—He was dull, evidently suffering from shock, as he did not take much notice of my examining him, whereas, when in good health, he greatly resented handling by strangers. He had eaten and drunk a fair quantity. The swelling over the ribs had not increased, but every five minutes he was seized with a clonic spasm, affecting the muscles of the near side, apparently from the head to the tail, causing that side of the body to curve slightly round, a slight groan being emitted at the same time. His attendant informed me the spasms had commenced shortly after I had left on the previous day, and had been present ever since, and that they were greatly aggravated by attempting to make him turn over in the box. I attributed these spasms to the effects of the blow on the near quarter, and thought

possibly some of the pelvic bones might be injured, but on considera-
tion I did not deem it advisable to make a rectal examination, as he
had defecated and micturated several times without difficulty, and
there were no signs of blood in urine or fæces.

Third Day.—His pulse and temperature were slightly raised, but
he ate and drank fairly well, passed several motions a little unnaturally
firm, and urinated without difficulty several times. The muscular
spasms were still present at intervals, and on auscultation over the slight-
enlargement over the ribs a distinct crepitus was noted during the
respiratory act. I therefore placed a small soft cushion over the
fractured ribs, retaining it in position by a wide roller bandage
applied sufficiently tight to restrict the movement of these ribs
during respiration, and gave strict orders against his being allowed
to lie down, and that nothing but laxative food be given.

Fifth Day.—The muscular spasms were not so frequent or the
crepitus so distinct; the bandage and compress were reapplied. He
was feeding nicely and apparently doing as well as one could expect;
pulsations 40, temperature $100\frac{2}{5}°$ F., ears and legs warm, though the
latter were slightly filled, probably from standing, bowels acting
regularly, and fæces of nice consistence. On taking him off the
rack chain I found that he walked very well with the injured side
next the wall of the box, but in the opposite direction he appeared
to roll behind. The same difference was noticed on turning him over
from side to side.

Sixth Day.—By request I visited him about 10 A.M., and learnt
that he had got loose and lain down during the previous night, but
I failed to detect any bad results from it.

Same day, 3 P.M., called in a hurry, as he was reported in pain,
which was attributed to his having eaten his bedding whilst loose, a
habit he was guilty of and for which he always wore a muzzle. His
pulse was 44, temperature $100\frac{4}{5}°$ F., conjunctival membranes of normal
colour, respirations slightly accelerated, extremities warm, no abdo-
minal distension, frequent scraping or pawing with first one and then
the other fore foot, but off fore mostly, occasional twisting of the tail,
had defecated four times since I saw him in the morning, one motion
of normal consistence and good quantity passed just previous to
my arrival, no signs of displacement of the fractured ribs, and no
crepitus or friction sounds detected on auscultation over them. I
learnt that the muscular spasms had not been so frequent that day,
that he had passed flatus several times and eaten all but his midday
feed. I gave morphia hypodermically and had the abdomen hand-
rubbed, and an hour later he was reported by the attendants as
free from pain and very eager for food, but I directed that none be
given for two hours, and then a small bran and linseed mash.

Midnight.—Reported again in pain although no food or water had
been taken. I found his pulsations 80, temperature 102° F., eyes very
staring, frequent sighing, conjunctival membrane slightly injected,
respirations accelerated and nostrils distended, ears cold, profuse
sweating, swaying towards the off side, head carried to one side,
continual wandering round the box, frequently partially losing his
balance behind, appeared nearly blind, abdomen distended superiorly.

Later.—Almost continual scraping with the off fore foot, no attempts
to lie down, no muscular spasms, breathing hurried, eyes full and star-

ing, conjunctival membrane not markedly injected, ears and legs rather cold, pendulous condition of penis, and no micturition since noon.

During the next twenty-four hours the following were the prominent symptoms :—

Pulse continued about 80 and small. Internal temperature varied between 101 and 102° F. Extremities cold, but not deathly. Sweating was profuse at intervals all over the body. No natural passage of fæces ; the enemas were retained for some time, and when ejected were slightly stained, and contained some shreds of mucus and one or two small hardened mucus-coated pellets. There had been no natural action of the bowels since 3 P.M. on previous day.

Pain was continuous, but no marked looking round to his abdomen ; he occasionally did look partly round, but it appeared to me to be more from anxiety than any pointing to the abdomen. He frequently wandered round the box, but this was a habit he often carried out for hours at a time when in good health. At intervals he threw himself down in a violent manner in spite of the efforts of his two attendants, one at each side with a halter. Sighing was a prominent symptom. Abdomen was not tympanitic, but appeared full superiorly. Borborygmus was occasionally heard, but very feeble. Muscular tremors and shaking of the whole body were present at intervals. There was no cough or nasal discharge, and the respirations were not materially increased. At times he breathed heavily for a few minutes. No external increase in the slight swelling over the injured ribs, or signs of depression of the fractured portions, and no crepitus and but slight friction sounds on auscultation over them. He drank some water once. Some two hours before death, after throwing himself down in a violent manner, he exhibited laboured respiration, gradual failing pulse, blanching of the mucous membranes, cold extremities, staggering rolling gait, boring of head, leaning against sides of box, tumbling forward, and on falling down was quickly dead.

Post-mortem.—Eighteen hours after death, showed rigor mortis well marked, body but slightly tympanitic, penis pendulous. Very little subcutaneous congestion, extensive recent bruising (but little external swelling) over middle of the eighth and ninth ribs on near side ; also bruise over the iliac spine on the same side. The stomach, spleen, liver, kidneys, bladder, and intestines were normal. The small intestines exhibited congestion of the mucous membrane, and contained bloody water. The cæcum was affected with marked muco-enteritis, as was also the large colon, but in a lesser degree. The floating colon contained mucus-coated fæces ; the iliac spine on the near side was not fractured, but the sacro-iliac articulation was injured. On removing the near fore leg and opening the chest, it was found to contain a large quantity of blood. The eighth and ninth ribs were fractured under the bruise, and three ribs were broken off against the spine and two against the sternum, the two latter fractures apparently quite recent, with a quantity of blood infiltrated into the tissues against the lower fracture. There was a large *post-mortem* clot in the near side, and fluid blood in both sides of the chest. The parietal pleura under the fracture of the eighth and ninth ribs was torn, and the visceral pleura of the same side was slightly damaged, but the lung tissue was not punctured. The right lung showed hypostatic

congestion. There was no clot in the right ventricle of the heart, and but a little frothy blood in the left. The pulmonary artery was ruptured (no doubt when he threw himself down), and this had been the immediate cause of death.

CASE No. 2.

One Tuesday evening in December last, I had a five-year-old brown mare brought in, said to have been ran into. I found a deep punctured wound in the front of the chest, just above the point of the off shoulder, and the animal was in a state of collapse, having been driven some distance since the accident. I syringed and plugged the wound and otherwise attended to the animal's comfort, and tied her head up to the rack to prevent her from lying down.

Friday.—Pulse was 60, temperature 104° F., appetite and defecations normal. A peculiar flapping and low grating sound was noticed, apparently coming from the supero-anterior part of the chest. There was œdema between the fore legs, and inability to extend the off fore limb, which was much swollen, slightly dropped at the elbow, bent at knee, and knuckled over at the fetlock. There was but little discharge from the wound, which was situated nearly 2 inches from the point of the shoulder, ran backwards between the scapula and thoracic wall, and also extended up the neck towards the spine. On account of the greatly increasing œdema the mare was allowed to lie down to-day, which she readily did on the near side.

Saturday.—The mare had regained the use of the off fore limb to a large extent. The œdema had not increased anteriorly, but posteriorly it had much increased, extending quite up the groin, where it was over an inch thick ; it ended along the sides of the abdomen in a well-defined line. The swelling was of normal warmth except at the most recent posterior portions, where it was a little warmer and more sensitive than natural. On pressure it pitted, had a doughy feel, and bloody serum exuded on tapping it. The wound was discharging clotted sanious matter, but the discharge was intermittent, and the clotted portion was more profuse after injection of the wound and movement of the limb, clearly indicating that it had been pocketed somewhere, although an effort had been made to give it exit through a depending opening. To this I attributed the greatly increasing œdematous swelling, which between the fore legs was nearly 3 inches thick, although it had been several times deeply lanced. The appetite was very good, there was no difficulty in feeding, and defecation was normal, but the neck was slightly twisted to the off side, and more or less fixed in that position. The haw did not show, and there was no erection of the tail or ears. She moved round towards the off side readily, and lay down for about two hours on the near side.

Sunday.—All the swelling of the off fore limb having subsided, she now used that limb as freely as its fellow. The edges of the wound were red and angry looking, and it was now discharging large quantities of pus, much like dirty curdled milk, and offensive in odour. When standing quietly by her off side, the peculiar noise in her chest could be plainly heard ; it recurred quite regularly, and was synchronous with every second respiratory act. The œdema had extended much posteriorly since yesterday ; the appetite was not quite so

good, in fact the neck was now so much twisted that she had to stand sideways—off side against the crib—to get her head in. The neck appeared slightly enlarged over the last three cervical vertebræ, where it was turned sharply round to the off side, and altogether it was much like what is seen in cows down in milk fever, only that the twist in this case was quite close to the anterior part of the chest, and the neck could not be extended into a straight line with ordinary force. She had not lain down that day.

Monday.—Pulse 50, temperature $100\frac{2}{5}°$ F., respirations slightly accelerated, very slight protrusion of the haw and erection of the tail, hind legs stuck out, abdomen tucked up, occasional muscular spasms, causing animal to nearly fall, appeared fully sensible and followed round the box, but always bearing to the off side, head and neck much more bent, in fact more than half-way round to the off side of the chest. She could turn the head half round on the neck, but could not move the neck, which appeared fixed as if dislocated at its lower third ; the muscles did not appear to be in any spasm on either side. The movements of the jaw were quite natural. The mare could pick and grind her food as if nothing the matter, and during the early part of the day she ate freely, but later she ate very little. The œdema was still more extensive between the fore legs, bowels acting fairly often, and fæces of nice consistence but slightly coated with mucus.

Monday Evening.—After eagerly drinking some chilled water from a pail held up to her head the mare was seized with a violent muscular spasm, which caused her to fall, and from this time all power of maintaining the normal position was lost. Whilst lying on the near side she was constantly struggling, and she was therefore shot.

Post-mortem.—On removing the skin it was found that all the œdematous parts were of a dirty reddish-grey colour, and had a most offensive odour. It appeared as if the subscapular tissues and muscles had been torn from the force of the blow on the surfaces of the ribs, after the shaft, which was that of a heavy spring cart, had entered the skin. The wound was about 2 inches wide, and ran upwards and backwards to the spine, where three ribs were broken off just below their heads, viz., the fourth, fifth, and sixth. The fifth dorsal vertebra was fractured and slightly displaced, and there was some discolouration on the inside of the chest under the fractured ribs and vertebra, but the pleura did not appear lacerated.

A CASE OF FRACTURE OF THE FEMUR IN AN ELEPHANT, SUCCESSFULLY TREATED.

By T. F. STANTON, D.V.A., Veterinary Surgeon to the Barnum and Bailey Show.

THE subject of this note was a young elephant belonging to Messrs Barnum and Bailey's Show. On 28th May 1897, the elephant fell while being unloaded from the cars at Clarksville, Tennessee, U.S.A. As she could not rise she was placed in a tarpaulin and carried by the men to the lot, a distance of half a mile or more, before I examined her, and was none too carefully handled. She was then conveyed on

a dray to my infirmary and placed in a sling, but at the end of five days I had to lower her on to the ground. She refused to stand on the uninjured leg and was doubled up in the sling, had stopped eating and would have died had she remained in the sling. For the next five weeks she was lowered on to the ground every night and raised again in the morning, and kept in the sling during the day. In six weeks she could stand on the injured leg, but I kept her in the sling every day, lowering her to the ground at night for another six weeks, and then sent her home. There was very little deformity or shortening of the injured limb (the femur shows 1½ inches shortening by actual measurement). The elephant had such perfect use of the limb, that, upon arrival at New York, two prominent veterinarians of that city who examined her reported to Mr Bailey that the femur had never been fractured.

The animal was destroyed at Olympia, London, 18th March 1898, because of repeated attacks of rheumatism, which effected both fore legs. The broken leg was never affected. The accompanying illustration reproduces a photograph of the animal's thigh bones.

The interesting features of this case are, (1) that the animal was carried over rough ground in a tarpaulin for over half a mile, and rather roughly handled before any attempt was made to reduce the fracture; (2) that it was impossible to keep her in a sling while the repair of the bone was in progress; and (3) that no splints of any kind were used, the limb being kept in position by leather straps alone.

As our lecturer on surgery in college pronounced this fracture incurable, and as Professor Williams expresses a similar opinion in his work on surgery, I thought the case was worth reporting.

Reports.

RECOMMENDATIONS OF THE ROYAL COMMISSION ON TUBERCULOSIS.[1]

MEAT.

A.—SLAUGHTER-HOUSES.

1. We recommend that in all towns and municipal boroughs in England and Wales, and in Ireland, powers be conferred on the authorities similar to those conferred on Scottish corporations and municipalities by the Burgh Police (Scotland) Act, 1892, viz. :—

(*a.*) When the Local Authority in any town or urban district in England and Wales and Ireland have provided a public slaughter-house, power be conferred on them to declare that no other place within the town or borough shall be used for slaughtering, except that a period of *three* years be allowed to the owners of existing registered private slaughter-houses to apply their premises to other purposes. The term of *three* years to date, in those places where adequate public slaughter-houses already exist, from the public announcement by the Local Authority that the use of such public slaughter-houses is obligatory, or, in those places where public slaughter-houses have not been erected, from the public announcement by the Local Authority that tenders for their erection have been accepted.

(*b.*) That Local Authorities be empowered to require all meat slaughtered elsewhere than in a public slaughter-house, and brought into the district for sale, to be taken to a place or places where such meat may be inspected ; and that Local Authorities be empowered to make a charge to cover the reasonable expenses attendant on such inspection.

(*c.*) That when a public slaughter-house has been established inspectors shall be engaged to inspect all animals immediately after slaughter, and stamp the joints of all carcases passed as sound.

2. It appears desirable that in London the provision of public in substitution for private slaughter-houses should be considered in respect to the needs of London as a whole, and in determining their positions regard must be had or the convenient conveyance of animals by railway from the markets beyond the limits of London, as well as from the Islington market, to the public slaughter-houses which should be provided. At the present time no administrative authority has statutory power authorising it to provide public slaughter-houses other than for the slaughter of foreign cattle at the port of debarcation.

3. With regard to slaughter-houses in rural districts, the case is not so easy to deal with. But the difficulty is one that must be faced, otherwise there will be a dangerous tendency to send unwholesome animals to be slaughtered and sold in small villages where they will escape inspection. We recommend,

[1] From the Report of the Royal Commission appointed to inquire into the administrative procedures for controlling danger to man through the use as food of the meat and milk of tuberculous animals. London : Eyre & Spottiswoode, 1898.

therefore, that in Great Britain the inspection of meat in rural districts be administered by the county councils. In Ireland the duty of carrying out inspection ought to devolve upon authorities corresponding as nearly as possible to those charged with that duty in England and Scotland. In view of the announced intention of the Government to introduce a new scheme of local government into Ireland we refrain from specifying the exact machinery which should be employed.

4. We recommend further. that it shall not be lawful to offer for sale the meat of any animal which has not been killed in a duly licensed slaughterhouse.

B.—QUALIFICATIONS OF MEAT INSPECTORS.

5. We recommend that in future no person be permitted to act as a meat inspector until he has passed a qualifying examination, before such authority as may be prescribed by the Local Government Board (or Board of Agriculture), on the following subjects :—

(*a.*) The law of meat inspection, and such byelaws. regulations, etc. as may be in force at the time he presents himself for examination.

(*b.*) The names and situations of the organs of the body.

(*c.*) Signs of health and disease in animals destined for food, both when alive and after slaughter.

(*d.*) The appearance and character of fresh meat, organs, fat, and blood, and the conditions rendering them, or preparations from them, fit or unfit for human food.

C.—TUBERCULOSIS IN ANIMALS INTENDED FOR FOOD.

6. We recommend that the Local Government Board be empowered to issue instructions from time to time for the guidance of meat inspectors, prescribing the degree of tubercular disease which, in the opinion of the Board, should cause a carcase, or part thereof, to be seized.

Pending the issue of such instructions we are of opinion that the following principles should be observed in the inspection of tuberculous carcases of cattle :

(*a.*) When there is miliary tuberculosis of both lungs . . . .

(*b.*) When tuberculous lesions are present on the pleura and peritoneum . . .

(*c.*) When tuberculous lesions are present in the muscular system, or in the lymphatic glands embedded in or between the muscles . . . . . .

(*d.*) When tuberculous lesions exist in any part of an emaciated carcase . . . .

} The entire carcase and all the organs may be seized.

(*a.*) When the lesions are confined to the lungs and the thoracic lymphatic glands . .

(*b.*) When the lesions are confined to the liver .

(*c.*) When the lesions are confined to the pharyngeal lymphatic glands . . . .

(*d.*) When the lesions are confined to any combination of the foregoing, but are collectively small in extent . . . .

} The carcase, if otherwise healthy, shall not be condemned, but every part of it containing tuberculous lesions shall be seized.

In view of the greater tendency to generalisation of tuberculosis in the pig, we consider that the presence of tubercular deposit in any degree should involve seizure of the whole carcase and of the organs.

In respect of foreign dead meat, seizure shall ensue in every case where the pleura has been "stripped."

MILK.

D.—DISEASES IN THE UDDERS OF COWS.

7. We recommend that notification of every disease in the udder shall be made compulsory, under penalty, on the owners of all cows, whether in private dairies or those of which the milk is offered for sale.

8. We recommend that for the purpose of excluding from their districts the milk of cows affected with tuberculosis of the udder, or exhibiting clinical symptoms of the disease, Local Authorities should be given powers somewhat similar to those of Sections 24-27 of the Glasgow Police (Amendment) Act, with power to slaughter such cows subject to compensation under the conditions named in the Report.

9. We also recommend that powers shall be given to Local Authorities to take samples and make analyses from time to time of the milk produced or sold in their districts, and that milk vendors shall be required to supply sufficient information as to the sources from which their milk is derived.

At ports where milk and milk products are received from foreign countries, any costs that may be thus incurred in their examination shall be borne by the importers.

E.—COWSHEDS, BYRES, ETC.

10. We recommend that the Local Government Board be empowered to require Local Authorities to adopt regulations as to dairies, cowsheds, etc., where that shall be found not to have been done already.

11. That in future no cowshed, byre, or shippon, other than those already registered, shall be permitted or registered in urban districts within 100 feet of any dwelling house; and that the discontinuance of any one already existing shall be ordered on the certificate, either of the medical officer of health that it is injurious to the health of human beings residing near it, or of the veterinary inspector that it is not a place wherein cows ought to be kept for the purpose of milk supply, and that it is incapable of being made so.

12. That the conditions of the attached cowsheds that shall warrant the registering of a dairy in a populous place, whether technically urban or rural, in the future shall include the following :—

1. An impervious floor.
2. A sufficient water supply for flushing.
3. Proper drainage.
4. A depôt for the manure at a sufficient distance from the byres.
5. A minimum cubic contents as regards such districts of from 600 to 800 feet for each adult beast varying according to the average weight of the animals.
6. A minimum floor space of 50 feet to each adult beast.
7. Sufficient light and ventilation.

While we have prescribed a minimum cubic contents and floor space without mentioning definite dimensions affecting ventilation and lighting, we are distinctly of opinion that these are by far the most important, and that requirements as to cubic and floor space are mainly of value as tending to facilitate adequate movement of air.

Existing cowsheds should be obliged to conform to the prescribed regulations within a period of twelve months from the time of the regulations coming into force.

13. The same conditions as those recommended for populous places should apply to cowsheds in sparsely populated places, except in so far as cubic contents per cow are concerned; as regards these cubic contents, such space per cow should be provided as would, in view of the surrounding circumstances, secure reasonable ventilation without draught. But the physical circumstances

prevailing in different localities being so various, we do not find it practicable to prescribe uniform minimum requirements in this respect.

14. We recommend that where cows housed in one district supply milk to another district, the Local Authority of the district in which the cows are housed shall be bound, when required, to supply to the Local Authority of the district in which the milk is sold or consumed full information and veterinary reports regarding the condition of the cows, byres, etc., whence the milk is drawn. Where the Local Authority of one district are dissatisfied with the reports so obtained, they may apply to the Local Government Board, with a view to an independent inspection and report being made.

F.—Elimination of Bovine Tuberculosis.

15. We recommend that funds be placed at the disposal of the Board of Agriculture in England and Scotland, and of the Veterinary Department of the Privy Council in Ireland, for the preparation of commercial tuberculin, and that stockowners be encouraged to test their animals by the offer of a gratuitous supply of tuberculin and the gratuitous services of a veterinary surgeon on certain conditions.

These conditions shall be—

(*a.*) That the test be applied by a veterinary surgeon.

(*b.*) That tuberculin be supplied only to such owners as will undertake to isolate reacting animals from healthy ones.

(*c.*) That the stock to be tested shall be kept under satisfactory sanitary conditions, and more especially that sufficient air space, ventilation, and light be provided in the buildings occupied by the animals.

16. We recommend that the Board of Agriculture in England and Scotland and the Veterinary Department of the Privy Council in Ireland undertake the circulation among agricultural societies of instructions for the proper use of the tuberculin test, with explanation of the significance of reaction, and directions for effective isolation of reacting animals.

SEVENTH INTERNATIONAL CONGRESS OF VETERINARY SURGEONS AT BADEN-BADEN, 1899.

In accordance with the resolution of the Sixth International Congress of Veterinary Surgeons, held at Berne in 1895, the seventh Congress will take place at Baden-Baden in the year 1899. The veterinary surgeons of Baden are entrusted with the carrying out of the arrangements. With the consent of an international meeting held at Stuttgart in June 1896, they have formed a committee of management, which has resolved to hold the Congress in Baden-Baden in the first half of August 1899.

The programme is as follows:—

(*a*) Precautionary measures against the spread of epidemic diseases in consequence of international trade in animals;

(*b*) The prevention of tuberculosis among domestic animals and the use of the flesh and milk of animals suffering from this disease, and, connected with this, the latest demands for an effectual meat inspection;

(*c*) The prevention of foot-and-mouth disease;

(*d*) The prevention of swine fever;

(*e*) The forwarding of veterinary science, especially by the erection of institutions for experiments in diseases, and by founding chairs of comparative medicine in colleges for veterinary surgeons;

(*f*) Conclusion of the work of drawing up of a common nomenclature in veterinary medicine;

(*g*) Official veterinarism.

This programme may be altered or supplemented if generally desired.

In the proceedings, besides the German language, English and French will be permitted. Arrangements will be made for the immediate translation of all speeches and reports.

In consideration of the great expenses connected with the Congress, the fee for members is fixed at M12 (= 12s). For ladies who wish to attend the congress, ladies' tickets will be issued on application, price M6 (= 6s).

Every member, even if unable personally to be present in Baden-Baden, will receive copies of all publications of the Congress, including the general report. The sale price of this report, which the members will receive free of charge, is fixed at M16.

Arrangements for rooms for members of the Congress will be made by a lodgings committee in Baden-Baden. The Committee is already in a position to state that those who take part in the Congress will be able to find board and lodging from M6 per day. The town of Baden-Baden has undertaken by arrangement with the Bad committee to provide special entertainments and festivities for the members.

The Grand-Ducal Government of Baden and the Chancellor of the Empire have generously made a considerable grant towards the expenses of the Congress.

Dr Lydtin, Geheimer Oberregierungsrath, Lichtenthalerstrasse, Baden-Baden, will be happy to give any further information which may be desired.

The Filiale der Rheinischen Creditbank in Baden-Baden will act as treasurer.

For information respecting lodgings apply to the Ortsausauschuss des VII. Internat. Thierärztl. Kongresses, Lichtenthalerstrasse, Baden-Baden.

The committee of management, in issuing invitations to the Congress, feels it may safely assure those who desire to take part in it that the time spent in Baden-Baden will not only be of the greatest professional importance, but will also offer the participants the pleasures and amusements of a first-class watering-place.

CONGRESS OF THE ROYAL INSTITUTE OF PUBLIC HEALTH.

18TH TO 23RD AUGUST 1898.

A Conference of veterinarians will be held in connection with the above Congress, at 10 A.M., on Friday, the 19th August 1898, in the Examination Hall of Trinity College, Dublin, with M. Hedley, Esq., F.R.C.V.S., Chief Veterinary Inspector, Irish Privy Council, as President.

Subject for discussion :—TUBERCULOSIS IN ITS RELATION TO PUBLIC HEALTH. Having regard to the far-reaching character of this subject, a series of papers bearing thereon will be brought forward for consideration.

It is hoped to obtain the co-operation of all important bodies, societies, and associations connected with the breeding, rearing, and general treatment of animals susceptible to the disease, as well as of those especially interested in the production of food, such as meat, milk, etc.

It has been decided, in connection with the Congress, to hold in all four sections and four conferences. There will also be a hygienic and bacteriological exhibition, held in the Royal University Buildings, from August 18th

to 27th. Besides the foregoing, a ball, given by the Lord Mayor, a banquet, at which the Lord Lieutenant will be present, various dramatic and musical entertainments and garden parties, also permission to visit places of interest in and around the City of Dublin, will constitute some of the hospitalities to be shown under the auspices of the Congress.

The Congress ceases on the opening day of the Horse Show, thus affording an opportunity of attending two important functions within a few days, and during one visit to the capital.

The minimum subscription for membership is 10s. 6d.

All communications relative to the Conference should be addressed to the Hon. Secretaries, Veterinary Conference, Harry Street, Dublin.
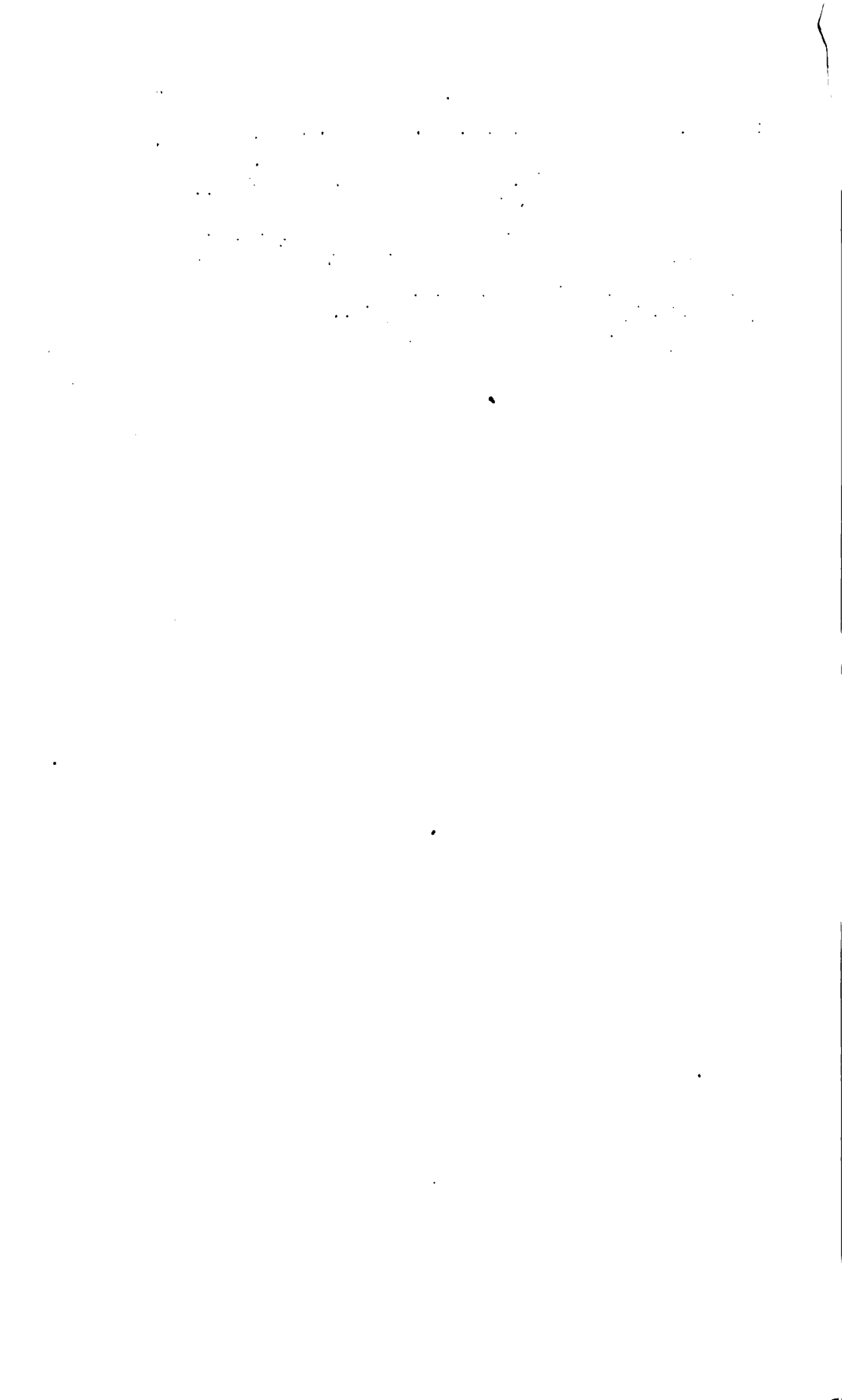
THE PATHOLOGY AND TREATMENT OF SPAVIN.[1]

By DR EBERLEIN, Berlin.

IN spite of numerous investigations the etiology and pathology of spavin have not yet been satisfactorily cleared up. The reason of this is possibly to be found in the fact that the majority of authors have contented themselves with attempts to improve the treatment of spavin, whereas the etiology of this disease has been neglected or altogether left out of account. This circumstance is all the more striking inasmuch as from the most remote times the veterinary surgeon and the breeder have had the greatest interest in spavin. The disease in fact possesses a striking economic importance, because on the one hand a very great number of horses suffer from it, and on the other hand because up to the present time the known methods of treatment suffice to cure only about one-half of the affected animals, and the uncured horses are more or less depreciated in value, since their fitness for work is in an important degree diminished by the disease.

At the instigation of my highly esteemed teacher, Professor Fröhner, as Repetitor of the surgical clinique here I began the following investigations in May 1895, and have continued them without interruption up till now. For this purpose I have had at my disposal the abundant clinical material and the accessory aids of the surgical clinique. Since October 1896, when the clinique for the larger domestic animals at this College was transferred to me, I have also been able to avail myself of the not inconsiderable amount of material furnished

[1] Translated from the "Monatshefte für Praktische Thierheilkunde." Band. XI. Heft 1. Enke, Stuttgart, 1897.

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by that department. Above everything else my attention has been directed to an elucidation of the pathology and treatment of spavin.

To Professor Fröhner I desire to express my sincere thanks for the continued interest which he has taken in my investigations.

LITERATURE.

In correspondence with the profound interest which spavin has at all times had for veterinary surgeons and horse-breeders, the literature of the disease is very great. It would greatly overstep the plan of this article to go minutely into the whole literature of the subject, and I shall therefore limit myself in general to reproducing the views of such articles as treat of the nature of spavin.

As early as the Middle Ages spavin was known to veterinary surgeons ; at least they designated by this name various diseases occurring in connection with the hock joint. As early as the middle of the thirteenth century Jordanus Ruffus (24) applied for this purpose the Latin term "spavenius," from which is probably derived the now common name of "Spat," which was introduced into the German language about the fifteenth century.

Marx Fugger (1), an experienced horse-breeder of the sixteenth century, also makes mention of spavin at several places in his well-known book on horse-breeding. He calls attention to the importance of the disease and the lameness determined by it, and specially warns against the purchase of such horses for breeding purposes.

V. Sind (2, p. 637), in his consideration of spavin, attaches most importance to the lameness and not to the bony enlargement. Further, he recognises that there is only one form of spavin, and says : " Since it more or less interferes with movement it has been divided accordingly into several varieties, for example, hock spavin, cock spavin, dry spavin, etc." The development of spavin V. Sind ascribes to over-exertion determining an accumulation of juices in the ligaments of the small bones, which eats away the parts and thereby brings about the production of callus.

An independent treatise regarding spavin, and one of much importance considering the period, was published by Busch (3) in 1788. This author discarded the designations blood spavin, bone spavin, hock spavin, and cock spavin, as referring to quite different diseases. He considered the division into visible and occult spavin as " laughable " (lächerlich), and that into dry and moist spavin as " brainless " (hirnlos). Busch referred the disease to a thickening of the lymph in the vessels of the ligaments, cartilages, and tendons of the joints, such thickening being produced by too long rest, extreme fatigue, sudden exposure to cold, etc. Busch further accurately described the bony new formations occurring in spavin, and recommended for treatment the use of Robertson's spavin ointment.

Kersting (4, p. 417) also rejects the subdivision of spavin into various forms, and says : " Among all these I recognise only one kind of spavin, whose quite distinctive character consists in the fact that it may at the one time be termed dry and at the other moist spavin." Kersting's description shows that by moist spavin he meant hygroma of the bursa of the flexor metatarsi, which develops suddenly and can run its course without lameness, whereas in dry spavin the

horse "goes lame for some time before the spavin makes its appearance as an elevation."

Blaine (5, p. 223) defines spavin as a bony outgrowth on the inner aspect of the tarsal bones, and he had observed it especially among horses with "cow hocks."

V. Arnim (6, p. 57) regarded rheumatism as the main cause of spavin, and he considered the cure of the disease extremely difficult or almost impossible. As the most certain assistance in diagnosis. this author recommended inspection and comparison of the inner contours of the hind limbs regarded both from the front and from behind.

Opinions regarding the nature of spavin suffered a complete revolution through the observation published by Havemann (7, p. 126) in the year 1822, namely, that in spavin the articular surfaces are themselves always diseased. Havemann therefore undoubtedly deserves the greatest credit for having been the first to point out disease of the articular surfaces in spavin. He says: "This (the cause of the lameness) always lies in the diseased articular surfaces. As soon as the articular surfaces of the flat bones become rough, and nature therefore attempts to bring about anchylosis of the bones, the lameness begins. In occult spavin the cartilaginous articular surfaces become dissolved and rough, and this makes the horse lame before the deposit of bony matter on the outside of the bones makes the spavin visible." Havemann further distinguishes hard or bone spavin, soft or moist spavin, and third, occult spavin.

V. Hochstetter (8, p. 79) includes under the term spavin all injuries of the hock which impede or weaken the movement and strength of the joint. With regard to the division of spavin into various forms, this author reverts to the older subdivision, and distinguishes blood spavin, bone spavin, and ox spavin. Bone spavin is ascribed by Hochstetter to tearing of the bone at the point of attachment of the flexor metatarsi, or to the "forcible tearing and swelling" of the atter.

Dieterichs, on the contrary, adopts Havemann's view, and defines spavin as a local disease of the hock joint of the horse, affecting the articular surfaces, articular cartilage, or bones. In his opinion the articular surfaces of one or other or several of the tarsal bones are always affected. Dieterichs also gives prominence to the occurrence of the so-called occult spavin, and says:—"Frequently the elevation is unimportant, scarcely visible or unobservable, and nevertheless the animal is decidedly lame. This is as a rule the case when the spavin is in the stage of development, as the articular surfaces are mainly affected, and have not yet become united." He recommended for the disease the technical term "Tarsarthrocace," which, however, has not been adopted.

E. F. Gurlt (10, p. 108) does not go minutely into the nature of spavin, but confines himself to the statement that in this disease "exostoses develop on the inner side of the hock, whereby fusion of the articular surfaces and ossification of the ligaments are almost invariably brought about."

In his lectures for horse-owners (11, p. 125) Hering gives a critical account of all the theories of spavin then known. In a general way he adopts the view of Havemann, but at the same time observes that

not seldom one finds the articular surfaces affected in horses that have not been lame, and that even in ordinary spavin the articular surfaces are frequently unaltered.

Havemann's theory of spavin found further support in the valuable investigations of G. W. Schrader (12). This author had found in his numerous examinations of diseased hock joints that the articular surfaces between the large and small cuneiform bones are as a rule the first affected. In his view this inflammation of the articular cartilage forms the starting-point of spavin, and "is succeeded by suppuration of the cartilaginous covering, and lastly by caries of the bones, which in a shorter or longer time is followed by union (anchylosis) of the diseased bones. In the ordinary process of spavin formation the exostoses so to speak mark the end. Generally they make their first appearance after the articular surfaces of the small bones have for a long time been highly diseased and caries has even attacked the outer edge of the scaphoid and large cuneiform bones." Schrader also calls attention to the fact that many horses without exostosis show decided spavin lameness, whereas others with the same show little or no lameness. The descriptions of this author are accompanied by two excellent illustrations.

That the external character of the hock joint by itself does not afford sufficient grounds for determining the presence or absence of spavin, Schrader (13) had previously pointed out. In an English thoroughbred with so-called "rough" (scharf abgesetzten) hock joints he had found the tarsal bones absolutely sound, whereas in an English half-bred horse without any trace of outward spavin enlargement he found that the large and small cuneiform bones were anchylosed.

In an article dealing with the subject Träger (14) corroborates the before-mentioned observation of Schrader. From an etiological point of view this author regarded spavin as "solely the expression of a gouty diathesis," whereas in this connection he attached only slight importance to great exertion and especially to racing.

Bartels (15), in opposition to the earlier authors, proclaims the "simplicity of the disease." He also expresses himself in opposition to Havemanns' theory, since he is of opinion "that originally spavin is an inflammation of the tendinous connections of the tarsal bones." He also discards the division into visible and occult spavin, and suggests for these the designations tarsal spavin and scaphoid spavin. In his view the former has its seat in the upper part of the joint and causes permanent lameness, whereas the latter is found in the neighbourhood of the scaphoid bone and the lameness determined by its presence is diminished on movement of the animal.

Strauss also (16, p. 606) does not recognise the term occult spavin, and he holds that Havemann's theory regarding the importance of disease of the bones and joints is not free from doubt. In the same way Hertwig (17) expresses doubts regarding the view that the lameness is caused by disease of the articular surfaces. He cites in opposition to this that he has often seen rough articular surfaces in his dissections of the hocks of horses which had never shown spavin lameness. On the contrary, he had in the anatomical examination of such joints frequently "found the bones abnormally red, more vascular and more porous, the periosteum somewhat thickened, and between it and the bones some coagulable fluid."

Bouley (37), however, in a lecture expresses himself in favour of disease of the articular surfaces as the essential factor in spavin. He says, " L'éparvin n'est pas seulement une tumeur extérieure située sur la marge des articulations des os plats du tarse, c'est plus que cela, c'est l'expression constant d'une lésion intérieure des articulations de ces os entre eux."

In like manner E. Gurlt (18, p. 623) describes spavin of the horse as esentially disease of the articular surfaces of the tarsal bones, and in his description he relies mainly on the observations of G. W. Schrader.

In their well-known work Friedrich and Carl Günther (19), notwithstanding that the subject of spavin is exhaustively treated, do not express any proper view of their own regarding the origin of this disease. They regard the nature of the morbid process in spavin as not yet sufficiently determined, and they do not accept as true either Havemann's theory according to which the cause of the lameness in spavin should be sought in the articular surfaces, nor the other theories according to which spavin ought to be regarded as an inflammation of the bones of the tarsus. On the other hand, these investigators say, however (p. 355) : " Spavin lameness without spavin enlargement, or the so-called occult spavin, depends on pathological changes which have involved the cartilages and bones of the tarsus, such as discolouration of the cartilage, development of points of bone on the affected articular surfaces, atrophy of the cartilage, and porosity of the bones. In these characteristic pathological factors of occult spavin resides the only cause of spavin lameness." Thus F. and K. Günther also recognise an arthritis or ostitis as the cause of spavin.

The literature of spavin was further enriched by the very exhaustive work of O. F. W. Schrader, jun. (20), entitled, " The Chronic Diseases of the Joints of the Horse." This author examined with great intelligence numerous hock joints from horses which he had treated for spavin during life, and he had thus seen everything that may be observed without microscopic examination. He specially describes with great accuracy the changes which occur in the cartilages of the articular surfaces. He says, " At the very outset of the disease we see on the articular surfaces, at a distance of one or two lines from the inner and anterior margin of the joint, one or several excavations in the bones. These are one or several lines broad, about as deep as the thickness of a playing card, and only partially filled and covered by a yellow gelatinous material formed from the altered cartilage. The excavation runs round the outer edge of the articular surface and encloses the central cartilage like an island." From this point the process spreads outwardly and inwardly and leads to anchylosis or the formation of osteophytes. Schrader designates spavin as a chronic disease of the joint, but does not give any definite explanation of its proper nature, and leaves the three following possibilities undecided :—

1. " The process begins in the cartilage, which is "weakened " or destroyed by the action of strong and repeated injurious influences, such as bruising, and it then spreads to the bones."

2. " The synovial membrane is first affected, and secretes too little synovia, or synovia of a morbid character, whereby the cartilage becomes diseased."

3. "The surface of the articular ends of the bone is first thrown into a diseased condition by repeated concussion, bruising, etc., whereby the capsule has its connections with the bone loosened, is deprived of its nourishment, and loses its vitality."

As we shall hereafterwards see, Schrader, jun. came very near to giving a correct explanation of the etiology of spavin.

Contrary to Schrader's views, Anacker (21) alleges that the cause of spavin lies in a chronic arthritis deformans proliferans. This author erroneously refers the degeneration of the cartilage to "a regressive amyloid process probably caused by the irritation of osteoid excrescences."

Roloff (22), like V. Hochstetter, Bartel, and others, ascribes spavin to periostitis determined by laceration of the tendons and articular ligaments, the periostitis as a rule beginning at the point of insertion. As a second necessary factor this author further recognises an affection of the bone determined by mechanical influences.

This view has been opposed by Schütz (23) in his well-known work regarding rachitis of the dog. He says, "just as little do I agree with the authors named when they say that spavin, etc., in horses proceeds from an inflammatory affection of the points of insertion of ligaments. . . . We have in the case of every joint a subsynovial division of the periosteum. It is from this layer that the formation of hyperostosis proceeds, never in the first instance from the before-mentioned points of insertion. The hyperostoses owe their origin to an arthritis, which, precisely on account of this alteration, has received the name of deforming arthritis, or arthritis deformans." Schütz thus regards spavin as arthritis deformans which secondarily leads to hyperostoses.

Contrary to the previously accepted views was that of Dieckerhoff (24), which appeared in his exhaustive monograph on spavin published in 1875. According to this author (24, p. 35) "spavin is a complicated chronic inflammatory process which has its starting-point in the inner layer of the bursa belonging to the fan-shaped (inner) branch of the tendon of the flexor metatarsi, spreads from there to the joint capsule and the periosteum of the under section of the tarsus, and thereby induces a chronic inflammation of the synovial membrane, with softening and detachment of the articular cartilage and inflammation of the bone marrow. The inflammation is thus at its starting-point a periarthritis, which only at a later stage becomes complicated with an arthritis."

This author also gives a new subdivision of the different forms of the disease and distinguishes—

1. Bone spavin, which is distinguished by bony new formation and slight fibrous thickening.

2. Serous spavin, corresponding with the moist or soft spavin.

3. Fibrous spavin, or connective tissue spavin.

4. Traumatic spavin, which is determined by penetrating wounds of the synovial bursa.

In correspondence with his view that the disease proceeds from an inflammation of the bursa of the inner tendon of the flexor metatarsi, Dieckerhoff recommends operative opening of the bursa as the best method of treatment of spavin.

Stokfleth (25 and 26), who also examined numerous hock joints from

spavined horses, recognises two possible modes of origin of spavin, namely, (1) from the exterior, from the ligamentous apparatus, or (2) from the interior, from the articular surfaces. He believes that when the former is the case the capsule becomes the seat of a chronic inflammation and becomes united with the tarsal bones. The inflammation extends to the periosteum, and leads to the formation of a more or less thick layer of fibrous tissue, which gradually becomes ossified from the interior outwards. After the bony new formation has in a greater or less degree restricted the movements of the small articulations, atrophy of the articular cartilage and anchylosis of the articular surfaces set in. In the immense majority of cases, however, according to his description, spavin proceeds from within outwards, by a primary inflammation of the articular surfaces. In twenty-seven of the cases examined by Stokfleth the disease had its origin undoubtedly in the ligamentous apparatus in only nine cases, while in eighteen of them it proceeded from the articular surfaces. With regard to the origin of the latter form, this author entirely corroborates the observations of Schrader, jun.

Shortly afterwards there appeared an article by Gotti (27), unfortunately not well known, which records excellent etiological investigations. This author examined a large number of tarsal joints, the greater number of which came from horses in which he had during life observed the more important symptoms of spavin. Gotti deserves the greatest credit for having by his investigations first brought the proof that spavin as a rule has its starting-point in the bones of the tarsus themselves. He found that the process sets in primarily with an ordinary very chronic ostitis of the scaphoid, large cuneiform, and metatarsal bones, in the course of which there is brought about a dilatation of the Haversian vessels, and a disturbance of the nutrition connective tissue elements of the bones. The latter gradually become converted into plastic marrow cells (marrow new formation), and thus lead to decalcification of the bones. In the further course of the disease the ostitis leads to a chondritis, which is characterised by a slowly progressing inflammation of the articular cartilage, with active proliferation of the ground substance and degeneration of the same. In the inflammatory process of the bones Gotti could distinguish a period of destruction (periodo distruttivo), during which the marrow new formation has the tendency to slowly extend, and a period of regeneration (periodo di riparazione), during which the marrow new formation becomes converted into compact connective tissue or bone. Thus, according to Gotti, the second stage leads to an ostitis condensans. If the proliferative process of the bone reaches the articular surface, as is the case with especial frequency in the cuneiform bones and the metatarsus, true anchylosis can be brought about by the conversion of the marrow into bone tissue.

The osteophytic new formations are regarded by Gotti as a secondary symptom and one which may be absent. They are usually the consequence of articular inflammation, and they then develop when the changes have reached the edge of the articular surfaces. Gotti has never observed any essential alterations in the bursa of the internal branch of the flexor metatarsi. This author accompanies his article by five plates with admirable microscopic and macroscopic figures.

The correctness of Gotti's views above cited has been verified by Bayer (28). He found the same very accurate, and he therefore entirely endorses them. In his investigations Bayer further found hock joints in which only a few osteophytes covered the surface of the bones although within the bones and the joints there were very advanced alterations, for which he considers the customary term occult spavin quite justified.

Möller (29) defines spavin as arthritis chronica deformans tarsi, and says: "To-day there is no longer any doubt that spavin is a chronic deforming arthritis; the only question in dispute is whence the disease proceeds." With regard to the pathology of the disease, Möller adopts the views of Peters (43).

In contrast to the preceding, Pflug (30), like Roloff, attaches most importance to the bony new formations. These osteophytes in his opinion are the principal anatomical basis of what is usually termed spavin (bone spavin). He also considers the disease as undoubtedly of inflammatory origin (periarthritis ossificans), and not seldom complicated with inflammation of the hock joint (arthritis).

Hoffmann (31) accepted as correct the views of Dieckerhoff, both with regard to the nature of spavin and its etiology. A year later (32) this author put forward another view of the nature of the disease. He now explains it as "an inflammation in the bones, a hyperæmia which leads to rarefying ostitis as well as ultimately to lacunar formation," and as the most effectual method of treatment he advises the use of needle firing.

Höhne (33) arrives at conclusions which are somewhat peculiar He regards spavin as a complicated disease which has its seat not in the hock joint but in the first place in the stifle joint. He treated a horse affected with occult spavin to which subsequently a gonitis was superadded. Höhne adds that he has repeatedly made the same observation.

The article by Smith (34), "Some Joint Diseases of the Horse," deserves special attention. It reveals a careful study of the physiological and anatomical conditions and alterations. The author distinguishes an articular and a non-articular form of spavin, and he has not yet seen the originally non-articular form become articular. The latter variety (articular spavin) Smith regards as an incurable form of joint inflammation (articular disease). Although the macroscopic examination of the joints was very carefully carried out and described, there is no account of the microscopic examination or of the examination of the bones. This is the more to be regretted, as, in consequence, the author has overlooked the primary ostitis, and declared the disease of the articular cartilage to be a passive process.

Aronsohn (35), like Pflug, regards spavin as periostitis resulting from laceration of the inner tendon of the flexor metatarsi and of the short inner lateral ligament, and explains the arthritis of the small joints as a secondary condition.

Fröhner (36) has also verified and confirmed the investigations of Gotti. He therefore accepts Gotti's view of the subject.

The foregoing abstract of the literature of spavin lays no claim to completeness, as it was only my intention to repeat here what is most essential. To record here all the views that have been published would, in my opinion, scarcely be possible, on account of the extra-

ordinary number of them, and at any rate it would be entirely superfluous. In the first place, the descriptions of spavin, especially those in text-books regarding the exterior of the horse, are frequently taken from other works, and, in the second place, many articles deal only with clinical material.

MATERIAL AND METHODS OF INVESTIGATION.

Owing to the frequency of the disease the finding of material presents no special difficulty. I had at my service a large amount of it in the clinique of the High School here. I also obtained a large number of hock joints from spavined horses through the kindness of Herr Feicke, Police Veterinary Surgeon, to whom I here express my thanks. In this way I was enabled to extend my investigations to a very large number of hocks, of which I have examined over one hundred.

In the first place the joints were examined with regard to the size of the bony new formations by inspection of their exterior, and they were then freed from the skin and subjacent loose connective tissue. Hereafter followed the examination of the bursa of the inner branch of the flexor metatarsi and the entire ligamentous apparatus. I then proceeded to the opening of the joint itself. At the outset of my investigations I found that this was attended with some difficulty, firstly because the small tarsal joints are united with extraordinary firmness by interosseus ligaments, and in the second place because the opening had to be effected with great care in order not to wound the bony growth on the articular surfaces. Simultaneously I examined the fluid in the joints.

The further examination was both macroscopic and also especially microscopic, and it extended to the character of the articular cartilage, bones, and articular ligaments. The macroscopic examination, which extended to the surface of the bones and to their appearance on section, presented no special difficulty, but, on the other hand, the microscopic examination was not in all cases easy. The latter was made with fresh material, with slices of bone ground down, and with sections. In this connection I obtained great assistance from the directions which are given by Schaffer (38) in his " Methods of Histological Examination of Bone Tissue."

The examination of fresh bone tissue was carried out according to the directions of Volkmann (38), small particles of rarefied bone being broken off with forceps, teased with needles, and examined in glycerine. In the preparation of ground slices and sections, the bone was first cut into small pieces and then fixed and hardened. I have obtained good results by fixing in concentrated alcoholic sublimate solution, or in 10 per cent. formalin solution (10 parts of concentrated formaldehyde in 100 parts of water) and subsequent hardening in alcohol of gradually increasing concentration (39, p. 12). When necessary the fat was removed with xylol. As a rule the pieces were taken from the scaphoid and cuneiform bones in such a way that the cartilage was left on one side of them.

By means of a circular saw fixed in a turner's lathe the pieces of bone were afterwards cut into thin slices. As a rule the slices thus obtained were so thin that they only required a little grinding down. At the outset I used a hand saw to make the sections, but those

obtained in this way were decidedly thicker. The sections were afterwards fastened on cork by means of Canada balsam or sealing wax, and then made sufficiently thin by means of fine files or rubbing on a sharpening stone. Finally, they were further rubbed down and polished, first on the one side and then on the other, on plates of ground glass. I never employed any sort of polishing powder.

In the preparation of sections the pieces of bone were first decalcified, and for this purpose I have used various fluids. I have obtained the best results with V. Ebner's decalcifying fluid, and also with phloroglucin (39) :—

Hydrochloric acid .	.	.	.	5·0
Spirit .	.	.	.	500·0
Distilled water	.	.	.	100·0
Common salt .	.	.	.	0·5

and

Phloroglucin .	.	.	.	2·0

with Nitric acid . . . . . . 20·0
(carefully warmed)
Nitric acid (10 per cent.) . . . 200·0

After washing, the decalcified pieces were again hardened in alcohol, and either immediately cut with the freezing microtome or embedded in paraffin (with careful warming). For staining purposes I have used with excellent results borax carmine. Among other agents I have also frequently used hematoxylin (eosin-hematoxylin), neutral carmine, picrocarmine, Bismarck brown, and safranin. Frequently I stained pieces in the mass with borax carmine or picrocarmine. Preparations were clarified by laying them for at least twenty-four hours in bergamot oil ; for this purpose I did not find xylol specially advantageous. Finally the sections were mounted in xylol Canada balsam.

In order to make the canals and lacunæ of the bone distinctly visible I have obtained very good preparations by impregnating thin slices with anilin stains after the method of Zimmermann (40). The slices were placed in xylol to remove the fat, well dried, and then boiled in concentrated alcoholic solution of fuchsin or gentian violet. They were then laid on the arm of a pair of forceps so that both surfaces were still covered with the staining solution, and they were thus allowed to dry for two days. The superfluous staining material was carefully scraped off, and the slices were again rubbed down in xylol on a plate of ground glass.

PATHOLOGICAL ANATOMY.

As is evident from the account of the literature of the subject already given, the pathology of spavin has been very variously interpreted by different investigators. Putting aside the oldest view, that spavin is due to a thickening or stagnation of the lymph, we may take the following theories of spavin from the literature of the disease.

1. Spavin is produced by disease of the ligamentous apparatus or tendons of the hock joint—V. Hochstetter, Bartels, Roloff, Stockfleth (nine times out of twenty-seven), Pflug, Aronsohn.

2. It depends upon disease (chondritis, arthritis) of the articular

surfaces of the small joints—Havemann, Dieterichs, Hering, G. W. Schrader, Træger, Bouley, E. Gurlt, Schrader, jun., Stockfleth (eighteen times out of twenty-seven).

3. It consists in an arthritis chronica deformans—Anacker, Schütz, Möller, Smith.

4. It has its origin in an inflammation of the bursa of the inner branch of the flexor metatarsi—Dieckerhoff, Hoffmann.

5. It is of the nature of an ostitis with subsequent arthritis—Hertwig, F. and C. Günther, Gotti, Bayer, Fröhner.

It will thus be seen that the opinions of veterinary authors regarding the nature of spavin are very divided. In general they turn on the question : Does spavin develop from within outwards (excentric), or from without towards the interior of the joint (concentric)? As is the case in many other diseases, the nature of spavin can best be explained by *post-mortem* examination, and I shall therefore deal first with that.

When the hock of a horse that has been lame from spavin is divided vertically with the saw, macroscopic examination shows in the scaphoid and large cuneiform bones morbid changes to which Hertwig (17) and Günther (19) have called attention. These are observable in the earliest stages of the disease, long before any alterations are visible in the articular cartilage or in the outer parts of the joint. Whereas the normal bones show on section a yellowish colour, the divided hock bones of a spavined horse, according to the severity of the disease, show in the form of specks or spots, or sometimes diffused over the whole surface, an intense red colour, and, in contrast to the normal hard consistence, they are so soft as to yield to the pressure of the finger. In slight cases of the disease these changes are restricted to small areas of the single bones, and they are then easily overlooked unless repeated saw cuts be made. This red colour of the bones, which corresponds with the areas of inflammation, is generally found in both the scaphoid and the large cuneiform bone, and it is always first observable in the portions of these bones which correspond with the antero-internal part of the tarsus. Not rarely the red places show a slightly stellate arrangement, and as a rule they lie in the neighbourhood of the articular surfaces (less frequently central), with which they generally stand in connection. In a similar way the inflammation frequently extends outwards towards the side, and involves the surface of the bones. This extension is, as we shall see, of great importance in the production of erosion of the cartilage on the articular surfaces, and for the formation of osteophytes. Fig. 1 shows in the scaphoid and large cuneiform bones an inflammation in which one may at the same time recognise the extension to the articular surfaces and the involvement of the outer edges of the bones. It can be observed that where the inflammation has reached the articular surfaces it ends immediately under the cartilage. As the bones have not been completely freed from the parosteal tissue, the osteophytes, which I shall discuss later, are not shown.

In the further stages of the disease the cut surface of the bones shows in the red inflamed parts light, or not seldom reddish, and as a rule round or elongated, places, in which one can observe finer or coarser meshes and larger or smaller pores. At these places the bone is also softer. If anchylosis has already set in the cut surface of the

bones exhibits still more marked distinctions. In the red inflamed parts there lie streaks, stripes, or rounded areas, which are distinguished from the surrounding parts by their yellow colour and firm character (osteosclerosis, ostitis condensans). The above-described stages are not always so sharply distinguished from one another, for they frequently occur simultaneously and therefore often blend together.
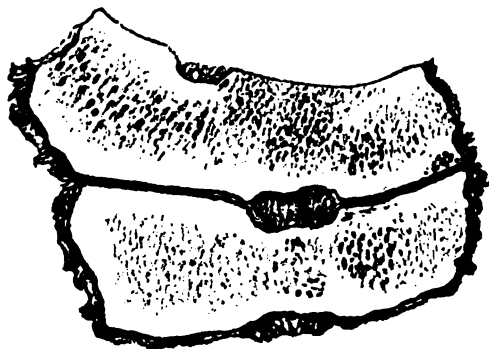
FIG. 1.
Tranverse section through the scaphoid and large cuneiform bones showing ostitis.
Natural size.

Where the inflammation has reached the articular surfaces we also observe changes taking place in the articular cartilage.

When such a joint is opened the first thing that strikes one is the diminished quantity of synovia. One finds only one or two drops of

FIG. 2.
Upper articular surface of large cuneiform bone showing punctiform erosion. Natural size.

joint-oil, without, however, any further abnormality. I have always found the reaction of the synovia to be alkaline, and the colour and consistence generally unaltered. When anchylosis has set in the synovia gradually becomes thicker in consistence and ultimately disappears. In fresh joints I have never found bacteria of any kind in the synovia, inflamed bone, or cartilage.

In the cartilage the first change observable is in the colour. In place of the normal whitish colour one sees at the diseased places a bluish or reddish colour. At the same time the shining aspect of the cartilage diminishes and is replaced by a dull appearance. Afterwards a superficial splitting into fibres sets in, followed by a deep fissuring
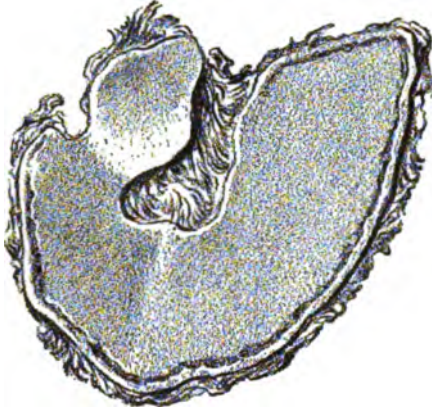
FIG. 3.

Upper articular surface of the scaphoid bone showing streaks of erosion. Natural size.

of the cartilage, which in the next place exhibits punctiform erosion (Fig. 2). As the disease advances the points become united by streak-like erosions.

The earliest changes are almost always found only 2-3 mm. from the anterior or inner edge of the tarsal bones, and as a rule they are
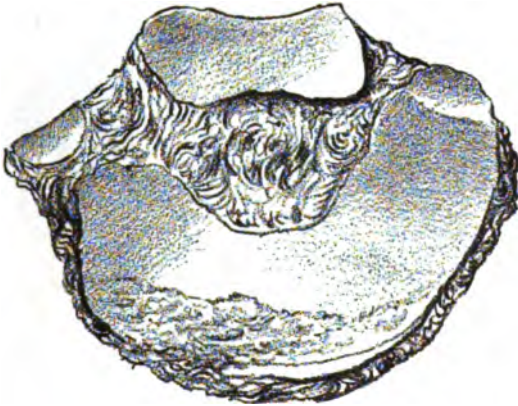
FIG. 4.

Articular surface of the large metatarsal bone showing erosion. Natural size.

first observable in the joint between the scaphoid and large cuneiform bones. Not rarely the other articular surfaces are first affected, but, as Schrader, jun. has correctly asserted, it is exceptional for erosion to commence in the centre of the bone. In those cases in which the

osteoporosis is quickly followed by ostitis condensans the cartilage does not always become fissured, but in place of that one then finds white punctiform projections of the size of a millet seed (calcified points). Similar alterations are found on the articular cartilage opposed to the diseased surfaces. In the further course of the
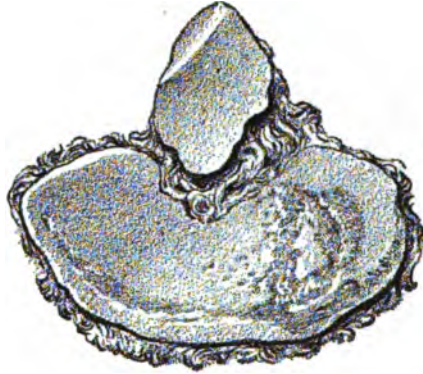
FIG. 5.

Upper articular surface of the large cuneiform bone showing erosion. Natural size.

disease the chondritis progresses, and spreads to the marginal part until the articular surface becomes surrounded by it like an island (Fig. 3). Before this or simultaneously the process spreads to the articular surface, and thus reaches the anterior or lateral edge of the same (Figs. 4 and 5). In consequence of the continual extension

FIG. 6.

Lower articular surface of the astragalus. ⅔ natural size.

of the chondritis there is gradually formed a flat erosion (Fig. 6), which involves smaller or larger portions of the articular surface (Fig. 7), and sometimes even the whole of that (Fig. 8). During these changes in the articular cartilage, which is not thereby completely destroyed but gradually split into fibres, numerous small bony projections are

formed on the surface by growth of the osseous trabeculæ, and give to the articular surface a markedly fissured appearance (Fig. 8).

The character of the morbid processes transpiring in the bone and cartilage can be with certainty made out by microscopic examination.
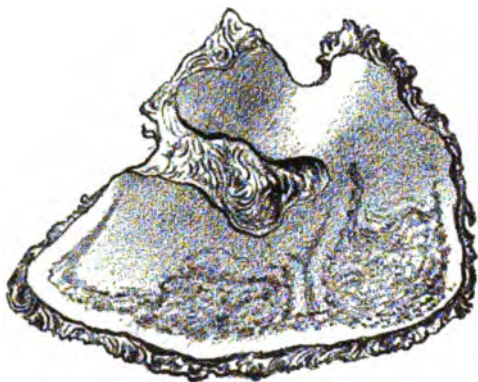
FIG. 7.
Lower articular surface of the scaphoid showing erosion. Natural size.

By that means, just as by macroscopic examination, it is found that the earliest alterations are in the bones.

When vertical sections are made through the before-described diseased bone tissue, one finds on examining the sections or ground slices with a medium magnification first a slight and later a steadily increasing dilatation of the Haversian canals. This is to be regarded as

FIG. 8.
Upper articular surface of the large cuneiform bone showing extensive erosion. ⅔ natural size.

an inflammatory process, which, as the microscopic examination of sections shows, develops as follows: In the earliest stages one observes in the Haversian canals an increase of the ordinary lymphoid marrow cells, which at first have a more marginal position. The number of these cells steadily increases, until the canals become completely filled by them and appear like granulating growths.

Simultaneously the bone tissue undergoes atrophy, whereby the width of the canals is increased. The Haversian canals, which in normal bone run mainly in the longitudinal direction and are connected by finely branched vascular canals, assume in consequence of these alterations a very irregular appearance, and form saccular dilatations at their points of division, so that a longitudinal section appears to be permeated by a labyrinth-like system of canals. The dilatation and filling of the canals with granulation tissue is also evident in transverse sections. Whereas in sound tissue the Haversian canals on transverse section appear as small point-like openings, they here take the form of large very irregularly shaped spaces. The absorption of the bone tissue leads to the formation of arched excavations and lacunæ (Howship's lacunæ) on the Haversian canals. Scattered between the marrow cells one finds many large multinucleated giant cells (osteoclasts), which lie directly on the bone tissue and determine its absorption. These, according to Von Kolliker's investigations (41, p. 25), are developed from osteoblasts. This inflammatory lacunar absorption attacks the surrounding bone in all directions, and leads to the formation of large cavities. On account of this peculiarity this type of ostitis is designated ostitis rarefaciens, or inflammatory osteoporosis.

Whereas at the outset of spavin only this osteoporosis is present, there soon sets in a new formation of osseous tissue, which is to be regarded as a regenerative osteosclerosis and compensates for the absorption of bone. The osteosclerosis, or ostitis condensans, by forming new bone substance reduces the size of, or completely fills up, the very porous osseous tissue, so that the bone not only regains its former consistence, but frequently becomes even firmer than before. In this process the newly formed bone substance is applied as osteoid tissue on the remaining osseous trabeculæ, and it is subsequently converted into firm bone tissue. Birch-Hirschfeld (41) is therefore right in regarding this osteosclerotic tissue as the equivalent of firm cicatricial tissue.

As I have already said above, the osteoporosis spreads indifferently in all directions, and involves the surrounding parts in the destructive process. In this way it arrives at the articular surfaces, where, however, it meets with resistance from the articular cartilage. The granulating new tissue here makes a halt, but it spreads out under the cartilage-covered surface, and thus interferes with the nourishment of the cartilage. As a result the upper strata of the cartilage became split into fibres, its deeper undergo softening, and these changes combined lead to a fissuring of it in a direction vertical to the articular surface. As a rule, however, there develops a pure chondritis, in the form of a progressive and regressive inflammatory process. The inflammation of the bone then spreads directly to the cartilage, and sets up in the latter an inflammatory proliferation of its cellular elements. On microscopic examination one finds that the ordinary cartilage cells, which lie together in clusters, are notably increased in numbers, and between them are scattered roundish or longish, large, nucleated cells or giant cells. The subsequent degeneration and resorption of the newly formed cellular elements lead to a loosening of the cartilage and a splitting of it into fibres, while at the same time all the strata of the ground substance become degenerated and fissured. The com-

pensatory condensation of tissue which sets in in the bone does not take place in the cartilage. The degeneration thus steadily advances and ultimately leads to a circumscribed or complete destruction of the entire cartilaginous layer. Outwardly these alterations make themselves apparent on the surfaces of the bones in the manner shown in Figs. 2-8.

In opposition to Gotti, I have repeatedly been able to observe that the granulating tissue which accompanies the osteoporosis penetrates the cartilage, that is to say, it grows into the cartilage, and breaks the latter down until it arrives at the articular surface. If now this is quickly followed by osteosclerosis a limit may be put to the chondritis, and there may remain on the articular surface only white, point-like projections, the so-called calcified points. Otherwise the chondritis quickly spreads, and leads to extensive destruction of the articular cartilage. My investigations therefore show that the
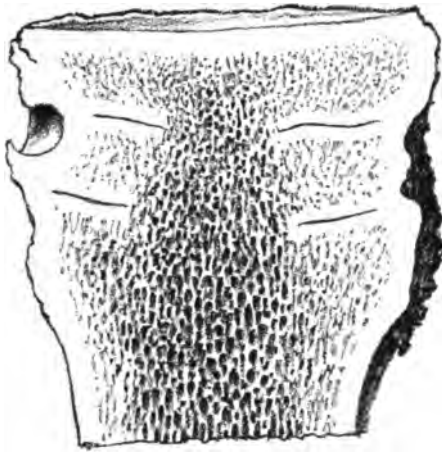
FIG. 9.
Vertical section through the scaphoid, large cuneiform, and upper end of the large metatarsal bones, showing anchylosis. Natural size.

alterations observable in the articular cartilages in spavin are ascribable to a chondritis which owes its origin to a primary ostitis.

In opening spavined hock joints one frequently observes that single bones, especially the scaphoid and large cuneiform, cannot be separated owing to their being united together by anchylosis. This takes the form of false or pseudo-anchylosis, or of bony anchylosis. The former is brought about by the parosteal hyperostoses which dovetail by means of finger-like processes with one another, whereas the osseous anchylosis takes its origin from the articular surfaces themselves. When a hock with anchylosed joints is divided by a vertical section one observes that the union of the articular surfaces has been effected only at some places, whereas at other places the joint is still recognisable as a cleft. In Fig. 9 the fusion of the articular surfaces is effected at the marginal part and in the centre, whereas the other parts of the articular surfaces are not united. If one breaks such a joint forcibly one finds, as Dieckerhoff (24) has

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specially pointed out, that the articular cartilage is still retained over more or fewer large areas, the union of the articular surfaces being thus only partial. As I was able to demonstrate, total anchylosis is a quite exceptional occurrence, whereas partial union of the articular surfaces is the rule. Judged by the nature of the intermediary uniting substance, the fully formed spavin anchylosis is to be regarded as bony. I have never observed a cartilaginous or fibrous union.

When a vertical section is made through an anchylosis of the scaphoid and large cuneiform bones one can make out by microscopic examination that the process develops in the following manner. When the articular cartilage is eroded the proliferating granulations of the Haversian canals are able to sprout out over the surface. As I have already pointed out, the erosion of the cartilage develops at the corresponding points of the opposed articular surface, and the granulating processes from the two surfaces meet and blend (anastomose), or they grow from the one surface directly into the tissue opposed to them. If now, in the course of the succeeding osteosclerosis, a new formation of bony tissue replaces the proliferation of marrow, the bones become united by the newly formed trabeculæ, and anchylosis is thereby effected. In this way the union may be either in the form of points or lines or have a flattened form. In the neighbourhood of the anchylosis one can distinctly follow the separate stages of the erosion of the cartilage, and make out how the splitting into fibres and ultimately the complete destruction of the same occurs. If this process develops less quickly, and the ossification sets in earlier, the bony new formations do not proceed directly from one bone into the other, but the proliferated bony trabeculæ of the one articular surface grow into excavations on the other, and become soldered together laterally by osseous matter. This form of anchylosis is less firm than the one first described. The articular surfaces when broken forcibly asunder show a rough fissured appearance (Fig. 8).

With this the process may come to an end, and through the occurrence of ostitis condensans and anchylosis a cure of the disease, or at least a disappearance of the lameness, may be brought about. In these cases little or no apparent enlargement makes its appearance on the exterior of the hock joint, and this form has therefore been designated "occult spavin." As a rule, however, in the course of spavin an enlargement shows itself at the inner or anterior surface of the hock joint ; this is to be regarded as the product of a periostitis ossificans, and it depends upon the formation of osteophytes. Several authors—Roloff, Pflug, Stockfleth, Aronsohn, and others, describe these osteophytes as the first alterations in spavin, and ascribe them to a laceration of the ligamentous apparatus. This, however, is not the case, and Schütz and others contradict such a view, and ascribe the development of the bony new formations to a preceding arthritis. According to my observations the development of the osteophytes is to be explained as follows.

As I have already shown, the erosion of the cartilage gradually spreads in all directions over the articular surface, and inasmuch as it generally begins only from 2 to 3 millimetres from the edge of the bones it very soon reaches the latter. As one can make out in micro-

scopic sections, the inflammatory process spreads from this point to the synovial membrane and the periosteum or subsynovial tissue, and
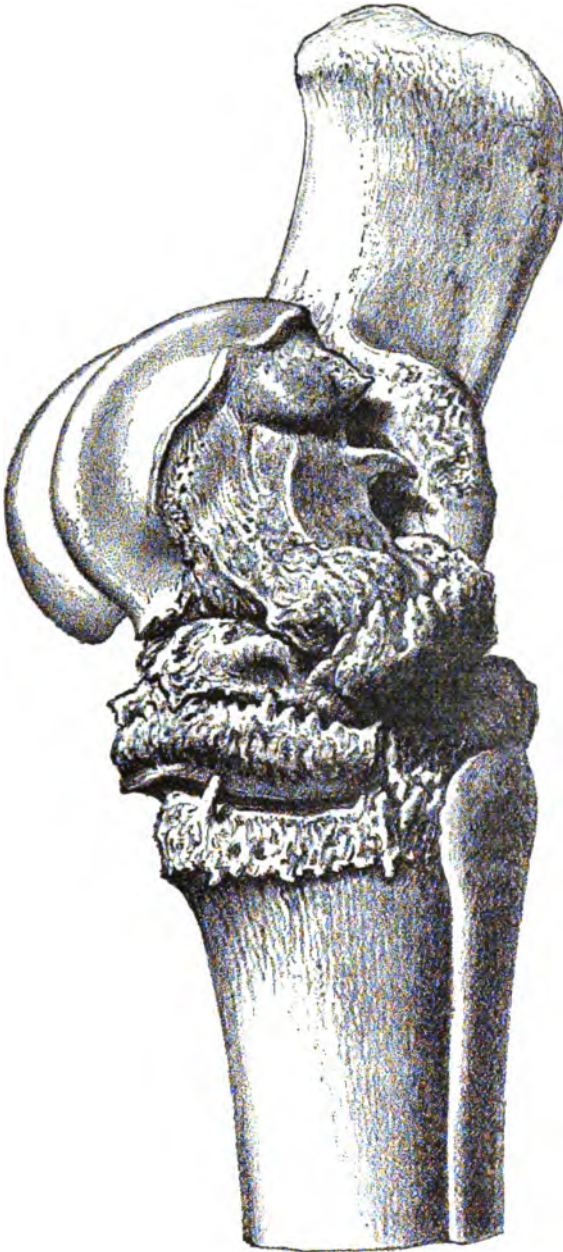
Fig. 10.

Right tarsus with extensive formation of hyperostoses on its inner surface. ½ natural size.

leads to the formation of hyperostoses.   I have in this matter been able to entirely corroborate the views of Schütz (23, p. 383), who says

" As is well known, the synovial membrane becomes attached where the cartilage ceases on the joint, and from this point the synovial membrane is united to the periosteum by subsynovial tissue. It is this layer that is the starting-point of hyperostosis formations, and these never have their origin from the points of insertion of the ligaments."

As a matter of fact, I have in no case observed that the hyperostoses took their origin from the points of insertion of the ligaments, nor have I ever seen spavin disease with hyperostoses and without arthritis. I, of course, make exception of the cases of traumatic periarthritis, which I shall discuss in connection with the differential diagnosis.

On the other hand, I have frequently observed that although osteophytes were already present the erosion of the cartilage had not reached the outer edge of the bone. In these cases the inflammation had extended direct from the bone to the periosteum. As Fig. 1 shows, the ostitis rarefaciens not infrequently spreads to the marginal part of the bone, and then directly sets up a periostitis,
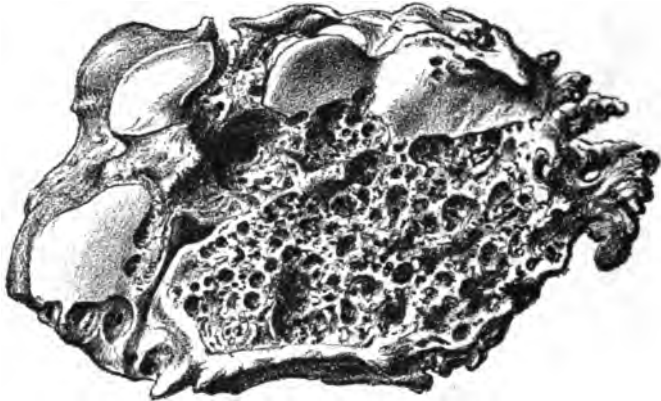
FIG. 11.

Upper articular surface of scaphoid bone showing orteoporosis and hyperostoses. Natural size.

which leads to the formation of osteophytes. This method of development may be accurately followed in microscopic preparations.

As regards the form of the osteophytes, these have been divided into the velvety, villous, tooth-like, laminate, styliform, etc. (41, p. 20). I have met with all these varieties in spavin, and indeed several forms at the same time in one preparation. They are specially distinct in microscopic preparations.

In all cases of hyperostoses the excavations caused by the various forms of processes are filled up with connective tissue, and the surfaces are thereby rendered comparatively smooth. In fresh preparations the small hyperostoses are on this account difficult to make out, whereas the larger are visible as a broad edge (Fig. 8). On this account also the osteophytes are not recognisable as such in Figs. 2-8. When, however, one macerates the hock joint or single bones of it (Figs. 10, 11, and 12), the smallest hyperostoses are distinctly visible. Fig. 11 shows bony new formations varying in size, form,

and direction, and at the same time it shows the fissured articular surface and the porous condition of the bones.
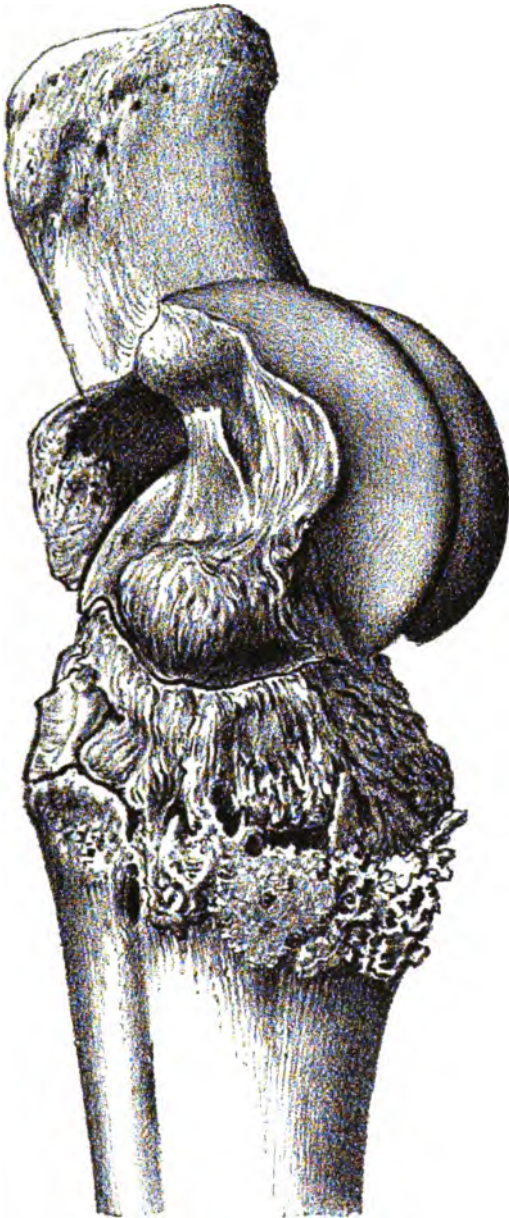
FIG. 12.

Left tarsus showing formation of hyperostoses on its flexor aspect and on the upper extremity of the large metatarsal bone, and anchylosis of the small articulations. ½ natural size.

Sometimes, especially in the case of the scaphoid and large cuneiform bones, the hyperostoses are hooked into one another, and thus

determine a stiffness of the joint which we designate pseudo-anchy-losis.

Finally, as regards the seat of the hyperostoses, these, like the other alterations, are frequently on the large and small cuneiform bones, and mainly on their inner aspect. Here (Fig. 10) the bony new forma-tions often attain a considerable size and show peculiar shapes. Furthermore, from this point they not infrequently spread out over the whole inner surface of the hock, as is partially seen in Fig. 10. As Fig. 12 shows, the first hyperostoses are often found at the upper edge of the metatarsus, where the inner passes into the anterior surface. As a rule this form is associated with a severe arthritis. I cannot confirm Gotti's observation that the formation of hyperostoses very often has its starting-point at the inner edge of the lower articular surface of the astragalus. On the contrary, I have observed that that part is seldom affected at the outset, but, as a rule, only at a very advanced stage of the disease.

I have never found striking appearances of disease in the parts surrounding or covering the small joints. As a rule the synovial membrane was thickened throughout, but it never showed newly formed processes. In the synovial fringes I have never been able to detect, either macroscopically or microscopically, morbid alterations. When anchylosis has taken place these fringes appear atrophied.

The ligaments of the joints—the inner lateral ligament and the short lateral ligament—showed nothing beyond a general thickening, except when there had been a marked formation of hyperostoses. In these cases the ligaments were frequently ossified in the neighbour-hood of their points of insertion. I have never observed splitting of the ligaments into fibres.

I have almost always found the bursa of the inner branch of the flexor metatarsi normal. In 120 hock joints which I have dissected I have only found the bursa diseased in two. In one of the cases the wall of the bursa was about twice its normal thickness. In the second joint the bursa was filled with a moderate quantity of yellow-ish fluid, and its wall showed spots of redness. But both the hocks showed at the same time older changes affecting the bones and the articular surfaces.

In only one joint did the inner branch of the flexor metatarsi itself show a slight splitting up into fibres; in all other cases it was sound. Where there had been a marked formation of hyperostoses there was almost always a smooth excavation for these tendons, and this afforded them sufficient accommodation.

The results of my anatomical investigations may be summed up by saying that they prove that in spavin the first change as a rule is an osteo-porosis—an ostitis rarefaciens affecting the scaphoid and cuneiform bones and the metatarsus, and very quickly followed by an osteosclerosis (ostitis condensans). In succession to the latter there develops in the affected articular cartilage a chondritis, with proliferation of cartilage cells and degeneration of the ground substance, and this leads sooner or later to anchylosis of the small joints. Frequently, but not in all cases, the inflammation then extends from the joint, or direct from the bone, to the periosteum of the small tarsal bones, and there sets up an ossifying periostitis, with the formation of hyperostoses on the

inner surface of the hock. The changes in the connective tissue surrounding the joint are of a secondary nature.

My investigations have thus confirmed the results obtained by Gotti, Bayer, and Fröhner. At the same time they have furnished the proof that spavin develops from within outwards, or excentrically, and they controvert the view of Roloff, Pflug, and Aronsohn, who ascribe to spavin a concentric development, that is to say, attribute it to a periostitis ossificans induced by laceration of the ligamentous apparatus, etc. If this view were correct one would find in all spavined hocks the first and oldest lesions in the form of hyperostoses, and one would also meet with diseased hocks without alterations in the bones and joints. I have often met with diseased hocks which showed advanced changes in the bony tissue and articular surfaces but little or no formation of hyperostoses, and I have never met with spavined hocks affected with periostitis ossificans which did not show an advanced ostitis and chondritis. I, of course, exclude the cases of traumatic periostitis (*see* differential diagnosis), which are sometimes erroneously classed with spavin.

The question which is now involuntarily forced upon us is : With what form of arthritis are the alterations in the articular surfaces to be classed ? Have we, as is generally accepted in veterinary litera-ture, to do with a disease corresponding with the chronic arthritis deformans of the human subject ? Schrader, jun., Gotti, and Fröhner declare themselves in opposition to this view, and as a matter of fact spavin of the horse for the following reasons cannot be identified with the disease mentioned. Chronic arthritis deformans of the human subject is a combination of degenerative and proliferative processes in the cartilage, bone, and capsular ligaments. The first disturbances begin in the cartilages (41, p. 60), lead to a sub-chondral inflammatory atrophy of bone, and excite marked prolifera-tive growth in the capsular ligaments. In contradistinction to this, spavin of the horse sets in with an ostitis, which is followed secondarily by a chondritis, and which never leads to proliferative processes in the joint capsule. In the two conditions the morbid process develops in an exactly contrary way, and hence spavin of the horse cannot be classed with chronic arthritis deformans.

Just as little can spavin be identified with the arthritis ulcerosa sicca of man. That disease is ushered in by nutritive disturbances in the cartilage, and these are accompanied or even preceded by nutritive disturbances in the bone substance. The morbid process is a regressive one, without the addition of intense proliferation of carti-lage or ossifying periostitis (41, p. 63). Spavin, on the other hand, is a proliferating ostitis, followed by chondritis and ossifying peri-ostitis, and it is therefore by its development to be distinguished from arthritis ulcerosa sicca.

Various authors have expressed the view that in spavin the symptoms of chronic deforming or ulcerative arthritis do not appear in a pronounced form merely on account of the stiff arrangement of the joints, and they therefore classify spavin with these joint diseases. That, however, is unjustifiable, for the separate classification of spavin is demanded not on account of the absence of one or several of the symptoms of the before-mentioned arthritis, but, above everything else, because of the mode in which the disease develops. Schrader

jun. (20, p. 154), therefore defines spavin as "a peculiar independent joint disease." I also regard the disease of the articular surfaces which accompanies spavin as a peculiar chronic arthritis which does not correspond either with chronic arthritis deformans or with ulcerative arthritis.

DEFINITION.

For the reasons previously discussed, and as the result of my anatomical investigations, I define spavin as a primary ostitis rarefaciens and condensans of the tarsal bones, especially of the scaphoid, large cuneiform, and metatarsal bones, followed secondarily by a peculiar chronic arthritis of the small tarsal joints, chiefly of the inner and distal joints, and (possibly) by a periostitis ossificans with the formation of hyperostoses on the inner and anterior surface of the tarsus, these again involving chiefly the scaphoid, large cuneiform, and metatarsal bones.

I will not deny that occasionally spavin may arise in another way, as, for example, in consequence of laceration of the ligamentous apparatus, etc., or as a consequence of a traumatic injury ; but these cases are so highly exceptional that they need not be taken account of in the definition.

It is hardly necessary to say that under the term "spavin" I understand only the so-called bone spavin. I exclude therefrom other diseases occurring in connection with the hock joints, such as the so-called blood spavin, serous spavin, connective tissue spavin, and traumatic spavin, because these are wrongly so named. As I shall show more fully in dealing with differential diagnosis, these conditions ought to be kept separate from spavin on account both of their anatomical alterations and their etiology.

ETIOLOGY.

In the etiology of inflammation in the osseous system there have generally to be taken into consideration—(1) a direct, locally acting injury, or (2) an injurious agent capable of exciting inflammation, and carried into the part by way of the blood stream. In either of these cases the cause may be infectious or non-infectious. In my investigations I have never detected an infectious cause, nor have I ever observed a hæmatogenous origin of the inflammation. Hence, in my view of the subject, the primary ostitis in spavin of the horse is always the result of a direct, locally acting, mechanical injury. Bruising of the small bones of the tarsus is thus the common cause of the development of spavin.

Unfortunately the opportunity for the production of such bruising is in various ways too often presented. For the practical consideration of the subject there is therefore a decided advantage in further subdividing the causal factors into a *causa externa* and a *causa interna*. As *causa externa* there is above everything else over-exertion of the hock joint, which may be the result of too prolonged or too rapid movement—kicking, jumping, quick turning, frequent covering in breeding stallions, heavy draught up-hill, working in deep land, etc. Repeated slipping in drawing heavy loads and bad shoeing

may also act in the same way. Klemm (42) has experimentally shown that excessive cutting away of the heels in shoeing is specially injurious in this way. Out of fifteen horses that had to be shod with low heels nine became affected with spavin in from one to two months. I have myself had the opportunity to verify Klemm's views, and have obtained the same results. Peters (43) observed that spavin was specially frequent on soft boggy land and uneven pavement. This he ascribes to the circumstance that in these conditions the twisting movement of the limb on the hoof, which is to some extent necessary, is hindered and transmitted to the hock joint.

The *causa interna* consists in an inherent predisposition depending upon the structure and internal arrangement of the hock joint, and of the whole limb, as well as upon the temperament and age of the horse. Even in the normally built hock joint we can recognise a special predisposition to mechanical lesions in consequence of its complicated construction, and this is naturally greater in hocks with a defective conformation. As objectionable forms of joint, in relation to spavin, are to be reckoned—the weak, the small, the flat, the thin, and the contracted (geschnürte) hock. I have frequently seen the so-called occult spavin developed in the flat and thin forms.

The consequences of defective conformation of the hock joint become more apparent when they are complicated by defective position. Slight departures from the normal position of the hock joint have no great importance for the development of spavin, but, on the contrary, the bandy-legged, cow-hocked, and wide positions favour the development of spavin. Peters (43) ascribes to the last of these types of conformation a special predisposition to spavin. The conformation of the hind-quarters and body is also of importance for the development of spavin. Observation teaches that horses with very strong pelvic muscles frequently become affected, especially when the legs are not of corresponding build. In this case, too, there is put on the firmness of the hock too great a strain, to which the joint is not equal. The frequent occurrence of spavin in heavy-bodied or long-backed horses is to be explained in the same way.

As early as the year 1770 V. Sind (2, p. 637) had pointed out that high-spirited horses are more liable to spavin than others. He says: "Spavin is a disease to which very many horses are subject; but it attacks especially those of high spirit, which throw energy into all their work." Nor is any explanation required of the fact that young horses more easily become diseased than old ones.

But the foregoing considerations do not in any way explain precisely why the inner side of the hock joint is so frequently attacked. Light is thrown on this point by the investigations of Hering (11, p. 155), who has ascertained "that the total body weight always acts more towards the middle line, and hence the inner parts of the limbs in many positions and movements have also more to carry than the outer." To that has to be added the fact to which Prosch first called attention, that in consequence of the screw-like form of the articular ridge of the astragalus "the foot during extension moves a little inwards towards the mesial plane." The pressure, especially during extension, is hereby conducted more to the inner side of the joint. I can, as the result of my own observations, merely confirm the views of Hering and Prosch, and also recognise in the before-

mentioned facts the cause of the frequent disease of the inner side of the hock.

As I have already mentioned, a further striking fact in the anatomical examination of diseased joints is that the erosion of the articular surfaces first shows itself some millimetres from the outer edge of the bones. According to my observations, the cause of this lies in the form of the articular surfaces. If one dissects away from a hock joint the surrounding connective tissue and the capsular ligament as far as the lateral ligaments, and applies pressure with the hands, one can observe that the closing edge of the small arthrodial joints of the tarsus does not coincide with the outer edge of the bones, but lies some millimetres (as a rule 2 to 3 mm., but the figures vary) within the latter. We have thus in these joints a condition similar to that which obtains in the cardiac valves—viz., a line of closure which does not correspond with the outer edge. Every mechanical lesion affecting the tarsus will thus naturally act most strongly on this contact margin, so that the first inflammatory changes must appear on the corresponding portions of bone and cartilage, as is in fact always the case.

SYMPTOMS.

The clinical symptoms of spavin are very many-sided, and the diagnosis of the disease is one of the most difficult tasks of veterinary practice. So true is this that Bayer (28) has remarked : " There is certainly no practitioner, however experienced, who is not perfectly conscious of having made a diagnostic error in one direction or the other." The difficulty of the diagnosis of spavin lies, above everything else, in the fact that spavin lameness occurs sometimes with and sometimes without spavin enlargement, and a spavin enlargement in like manner sometimes with and sometimes without lameness. Hence, in speaking of symptoms, one conveniently distinguishes between spavin lameness and spavin enlargement.

While at rest the horse generally holds the diseased limb with the joints flexed, and rests the foot on the toe. If both hind limbs are affected they are alternately used to support the weight and rested. By keeping the fore feet well backwards the horse also strives to throw the body weight as much as possible on these. If made to move sideways this is done without trouble towards the side of the diseased limb, but pain is evinced if the movement is in the opposite direction, because this throws more weight on the inner side of the diseased joint. In extreme degrees of the disease, however, this difference becomes less marked.

The lameness *per se* is not at all so characteristic that one may base the diagnosis "spavin" on it alone, and as a rule a certain diagnosis is first made possible by the functional disturbance in combination with the anatomical changes on the inner side of the hock joint. In all cases, however, by an accurate examination of the whole limb, it must first be ascertained that there are no other alterations to account for the lameness. This is especially necessary when there is no spavin enlargement—that is to say, in occult spavin.

The form which the functional disturbance takes is that the hoof is rested on the toe only, and a complete extension of the foot under the body weight is avoided. The extremity, when it leaves the

ground, is by sudden flexion carried forward with abnormal quickness, so that in severe cases of the disease the action is like that of stringhalt. In order to compensate for the shortening of the step which this entails, the hip joint is flexed to an extreme degree. One also observes a diminished flexion of the hock joint, in consequence of which the animal when on hard ground throws the weight off the heels and keeps them higher. In severe cases of the disease there is sometimes observable a position of abduction, which, when both limbs are affected, makes the animal appear strikingly wide behind in moving or standing. In walking many horses show an apparent lengthening of the step in front. In order to compensate for the diminished extension of the hock joint, the stifle is flexed to an abnormal degree, and the angle of the hip joint is opened by sinking of the pelvis towards the affected side. When there is very marked disturbance of action the horse often prefers galloping to trotting.

For the diagnosis and prognosis of the disease the occurrence and course of the lameness is far more important than the character of it. As a rule the lameness develops gradually, and at the outset of the disease it is observable only during the first steps of movement. In the later stages the lameness becomes more pronounced, and is seen especially on short turning. It almost always, however, retains the peculiarity already described by V. Sind (2) and Kersting (4), of disappearing on movement and reappearing after rest. Only in very rare cases does the lameness increase with exercise, and this symptom is seen only when the disease is very severe.

Various theories regarding the cause of the lameness have been advanced by veterinary authors. The older view, which ascribed the lameness to the pain caused by pressure of the exostoses on the periosteum, owes its origin to the rough character of the macerated bones. But inasmuch as during life the inequalities are made level and completely smooth and slippery by connective tissue, this theory must be considered erroneous. I have also been unable to convince myself of the correctness of Dieckerhoff's assertion that the lameness is ascribable to inflammation of the bursa of the flexor metatarsi. According to my observations, the lameness is ascribable to the presence of a rarefying ostitis and arthritis of the small joints of the tarsus, as is already indicated by the disappearance of the lameness when the osteoporosis has been abolished by the condensing ostitis, and the arthritis by anchylosis. I have repeatedly been able to verify this view of the question in the examination of hock joints of horses which had formerly suffered from spavin.

As a rule in spavin there is a bony enlargement on the inner aspect of the hock joint. As I have already explained, this is due to the preceding ostitis and arthritis, and it may be absent. In the latter case the disease is termed "occult spavin."[1] In respect of form, size, and situation, spavin enlargements show great variation (*see* Figs. 10 and 12). They are sometimes flat, sometimes projecting, sometimes scarcely observable, and sometimes visible at a distance. As regards their situation, they generally correspond with the scaphoid and cuneiform bones, but they are sometimes higher, sometimes lower, sometimes towards the front, sometimes towards the back. The large

[1 The literal rendering of the German term (unsichtlar) is invisible, but the word "occult," as applied to spavin by English veterinary surgeons, denotes the same condition. J. M'F.]

protuberances are very striking to the eye, but the detection of the smaller exostoses is sometimes attended with difficulty. With this object one allows the horse to stand squarely, and compares the contours of the joints. Enlargements lying well forward are best seen when the observer places himself near the animal's shoulder, first on the one side and then on the other. In all cases it is advisable to previously smooth down or wet the hair over the part. On palpation of a spavin enlargement one recognises a new formation of bony hardness and firmly united to the underlying parts, while the skin over it feels more firmly adherent and thinner than on the sound side. Symptoms of inflammation, such as warmth and sensitiveness, are very seldom present.

Spavin enlargements must not be confounded with those alterations of form which are exhibited in the so-called "course" (scharf abgesetzten) hock joints. The distinction is not always easy, for according to Schwarznecker's observations, which have been confirmed by Möller (45), and which I can corroborate from my own investigations, the hock joints are often built asymmetrically on their inner sides. The course hock is regarded by Möller as within certain limits a physiological compensation, and of course it exists without lameness. It is generally found at the posterior part of the inner face of the joint, never only in the neighbourhood of the flexor aspect, and by this more than anything else it is to be distinguished from spavin enlargement.

As a rule there sets in after a short time an atrophy of inaction affecting the muscles of the pelvis and leg, which is very obvious when opposite limbs are compared. This affects especially the pelvis, and it sometimes develops very quickly, notably in fat animals.

In my clinical examinations I have frequently met with an apparent diminution in size of diseased tarsal joints, which has not yet to my knowledge been described, although it has in my view considerable importance. I regard it as an example of atrophy caused by inaction, and it affects the entire joint. In order to obtain an accurate estimate I have made measurements by means of a string applied to the circumference of the diseased joint in its upper, middle, and lower parts, these being then read off on a centimetre scale. In order to avoid mistakes I took special care to apply the measure at exactly the same height in the different hocks, the lower string being always applied over the exostosis. My numerous measurements have shown that at the level of the upper and middle bands the sound hock was always thicker than the diseased one, and the difference amounted to 1-6 cm. The measurement made over the spavin enlargement of the diseased joint was as a rule only 1 cm. less than that of the sound hock, and frequently indeed it was equal to the latter; only in two cases was the circumference 1 cm. greater in the diseased hocks, and in these the spavin projection was large.

Ultimately, when the disease lasts a long time, deformities also set in in the hoof. The angle of the toe becomes more obtuse, the toe shorter, and the heels reach a striking height. The shoe is then worn away only at the toe.

In the examination of horses showing spavin lameness the considerations which have been discussed above must be kept in mind

in order to arrive at a correct diagnosis. To facilitate the same and render it more certain the spavin test may also be carried out. For this purpose the diseased leg is pushed upwards so as to flex all the joints as strongly as possible, and it is held in this position by the point of the toe for two or three minutes. The horse is then trotted, and after about ten or twenty paces he shows a more pronounced lameness than before. However, the spavin test is of value only when the examination of the limb in other ways has yielded negative results.

Lastly, as the results of the investigations by Pfeiffer and myself (46), I can recommend the employment of the Röntgen rays as an aid in diagnosis, especially in the so-called occult spavin. For information regarding the method of making the examination and the interpretation of the picture thus obtained I refer readers to our reports on the subject (46).

DIFFERENTIAL DIAGNOSIS.

Notwithstanding the complexity and instability of the symptoms it is still possible by an accurate examination to guard against errors. At the same time, disease of the hip joint, chronic gonitis, distorsion of the phalangeal joints, ring-bone, stringhalt, etc., may give rise to mistakes. As a rule it is possible to arrive at a correct decision by making an exact examination, and at the same time observing the maxim to make a positive diagnosis of spavin only when no other important alterations capable of causing lameness are present anywhere in the limb.

I will therefore here consider only a few of the diseases of the hock joint itself which are of importance from the point of view of differential diagnosis.

1. The so-called serous spavin, also termed spavin gall, moist or soft spavin, is a hygroma of the bursa of the flexor metatarsi, and has therefore nothing to do with spavin. Fluctuation may be made out in the bursa by palpation, and this is decisive.

2. In the so-called fibrous spavin, which consists in a fibrous thickening of the skin at this place, palpation likewise enables one to decide as to its nature.

3. The so-called traumatic spavin. By this term is falsely understood that general inflammation of the hock joint which owes its origin to a blow, kick, stab, or other similar mechanical injury, and which may run an infectious course or not. It represents a traumatic periarthritis, is for the following reasons not identical with spavin, and therefore ought not to be reckoned with the latter :—

(*a*) Periarthritis develops concentrically, spavin on the contrary excentrically.

(*b*) The former develops suddenly after a traumatic injury and with great swelling of the whole joint, whereas the latter develops gradually and with little or no local swelling.

(*c*) The former shows acute inflammatory symptoms (pain, heat), whereas the latter does not.

(*d*) The former often runs an infectious course, the latter never.

(*e*) The latter is always connected with disease of the bone tissue and articular surfaces, whereas that is generally not the case in the former.

I have dissected the hock joints of two horses that had been treated for periarthritis in the surgical clinique five or six weeks previously. In both cases I found the articular surfaces intact, although an extensive growth of osteophytes had taken place. Gotti (27, p. 22) has made a similar observation. That author says :—
" When one compares the whole series of spavin alterations commonly observed in the before-cited cases of tarsal inflammation, which begin in the peripheral tissue and are followed by extensive periostitis, one easily obtains the proof that the articular surfaces remain unaltered either for a long time or permanently, although frequently the richest formation of osteophytes has led to anchylosis (spurious) of the entire hock or of some of its articulations."
These observations show that, contrary to the view frequently accepted, periarthritis is not identical with spavin.

PROGNOSIS.

The prognosis in spavin is always uncertain, since it depends upon the nature of the animal's work, its age and breed, its conformation, the position of the hock joint and limb, the degree of the lameness, and the seat of the disease. From a practical point of view it is advantageous to distinguish between spavin enlargement and spavin lameness.

Inasmuch as we are not in a position to remove or materially diminish the spavin enlargement by therapeutic means, the prognosis with regard to that is in all cases unfavourable. It also remains doubtful when the enlargement is not accompanied by lameness, for according to experience the presence of the enlargement gives a permanent predisposition to spavin lameness. Even a spavin enlargement that has existed for years without lameness, especially if it is situated near the flexor aspect of the hock, may suddenly give rise to lameness when the limb suffers a mechanical injury or the animal has to undergo severe exertion. The unfavourable prognosis in spavin enlargement is also explainable when we reflect that the hyperostoses are the result of an arthritis, and that the size of the same is not at all in direct proportion to the degree of joint inflammation. As I have already remarked, the size of the enlargement is practically unaffected by therapeutic measures. After the use of a blister or firing a sclerosis of the skin frequently sets in as a result of the inflammation, and this, by making the sharp contour of the enlargement indistinct, deceives one into thinking that its size has been diminished. As regards their position, spavin enlargements are to be regarded the more unfavourably the further forward they are on the hock, that is to say, the nearer they are to the flexor surface. Indeed, although these forms are as a rule not very large I have observed that they are always associated with severe arthritis.

In practice, however, the important point is the prognosis of spavin lameness. This requires the greatest prudence, for, as we have already seen, the cessation of the lameness depends upon the reparative osteosclerosis and anchylosis of the small tarsal joints, and it is extraordinarily difficult to determine whether these have set in or not. On this account alone the prognosis of spavin lameness must be unfavourable or at least doubtful. In conformity with this view

stands the fact that veterinary surgeons have always given special prominence to the difficulty of curing spavin, some authors even holding, erroneously, however, that the disease is always incurable.

As Havemann (7) has already pointed out, spontaneous recovery from spavin lameness sometimes takes place, that is to say, the lameness completely disappears in the course of time. Such cases, however, are exceptional. In a large number of cases the lameness can be removed by appropriate treatment, and in others it can be notably diminished. At the same time, a not unimportant number of diseased horses remain uncured. To this class belong especially the subjects of so-called occult spavin. It is generally reckoned as the result of experience that about 50 per cent. of cases of the disease can be cured by the customary methods of treatment, and my own observations are in agreement with this estimate.

Sometimes spavin brings on other diseased conditions of the limb which render the cure more difficult or even impossible. Among such complications, upright pastern, or knuckling over at the fetlock, ringbone, inflammation and contraction of the flexor tendons, and disease of the stifle joint, are specially to be feared. Obviously, these complications render the prognosis decidedly more unfavourable. In such cases the horse generally remains lame for life, and either useless or fit only for the lightest kind of work. The prognosis is equally bad when during the course of the disease marked muscular atrophy and loss of appetite set in as the expression of extreme lameness and intense pain.

Lastly, a question of very great interest in horse-breeding is : Ought spavined horses to be used for breeding purposes or not? Quite correctly, but in contradiction to the earlier view of the subject, Dieckerhoff (24, p. 193) holds that with reference to this question every case of spavin must be judged on its merits. As I have shown in dealing with the etiology of the disease, the primary ostitis rarefaciens of the small tarsal bones is the result of a mechanical injury of the same (contusion, bruising), and it may therefore be brought about from external causes in the best formed hock and leg. Hence, the spavin itself is not hereditary. It is, however, scientifically proved that the defective conformation of the hock joint and the faulty position of the limbs which invest the horse with a predisposition to spavin (causa interna) are hereditary. From this it follows that, with respect to spavin, only such horses as have defective hock joints and faulty position of the limb on the one hand, and bad bony structure generally on the other, need be excluded from breeding.

THERAPEUTICS.

It need hardly be said that from the nature of spavin an absolute cure of the disease is not attainable, and that all therapeutic efforts can only aim at bringing about a relative cure of the disease, that is to say, a removal of the lameness. As I have already explained, this again can only be effected by the occurrence of osteosclerosis in the bone tissue, and anchylosis of the small joints of the hock. At all times certain veterinary surgeons have brought into use what they alleged to be infallible cures for spavin, and the multiplicity of opinions regarding the nature of the disease is reflected in the great

variety of the vaunted methods of cure. Some of the procedures recommended will not bear professional criticism. Reinbold, for example, can hardly have been serious when he proposed to bring about the necrosis and extrusion of the small cuneiform bone by blistering with euphorbium and cantharides.

Since the internal treatment of spavin has proved useless, we may turn at once to the surgical methods of treatment. Of these blistering and firing have from ancient times been held in greatest repute. It is claimed for these that they induce a speedier union between the articular surfaces of the small bones of the hock, change the inflammation from chronic to acute, and thereby cure the disease. Very various ointments and liquids are in use as blisters. Among the agents employed for this purpose are cantharides, euphorbium, croton oil, red iodide of mercury, corrosive sublimate, arsenic, etc. (compare the text-books of special surgery). Care must be taken not to rub the blister into the flexor surface of the hock joint, as otherwise, in consequence of the inflammation, changes which are not easily combated may be set up in the skin. Notwithstanding numerous trials, I have not yet been able to convince myself of the good effect of blistering. In order to test their value in spavin I have for a considerable period in my clinique treated one-half of the cases of spavin by firing and the other half by blistering. The latter were all treated under my care and with various medicaments, and in several of them distance firing was also practised on the blistered part. The result was that of the eighty horses thus treated only 10, or $12\frac{1}{2}$ per cent., were cured, while the remaining cases had to be subjected to other methods of treatment. Better results might possibly be obtained by the subcutaneous application of blisters recommended by Busch (3) and more recently by Bassi (50). The latter makes three converging incisions through the skin of the diseased part, loosens the subcutis, and then thoroughly rubs in the blister.

Of all methods of treatment the firing iron is the one that ought to be most frequently employed, either in the form of line or point-firing. According to Busch and others, the first of these was formerly often used, and especially in England as a prophylactic. At the present time line-firing ought only rarely to be used for spavin. Of point-firing, the cutaneous, percutaneous, and perforating methods are in use. To cutaneous point-firing, which is carried out with a pear-shaped iron, one cannot ascribe any special curative action. Percutaneous point-firing penetrates through the skin, and can therefore act directly on the periosteum and the periarticular tissue. For this purpose an iron with a sharp point is used. In practice this method has a great reputation. In perforating firing the firing iron penetrates into the bone tissue itself, and it there exerts an intense action. I shall describe this procedure at length at the end of this article. Lastly, Botazzi (48) has recommended the subcutaneous application of the firing iron. He casts the horse with the diseased leg undermost, clips the hair off the spavin enlargement, and makes over the centre of the latter a 4-5 cm. long incision through the skin. While the lips of the wound are held apart he removes the subcutaneous tissue and burns two or three points in a triangle with its base upwards. Antiseptic dressing. It is said that the horse is again fit for work after thirty days.

The insertion of a seton on the inner aspect of the hock had at one time a great reputation in the treatment of spavin, and Dieterichs and Hertwig especially have dwelt upon it, but it is now completely fallen into disuse. The seton, which was left in for two to three weeks, was pulled round daily and smeared from time to time with some irritant material. The horse must be cast for the operation.

Various other operations have been recommended for the cure of spavin. Abildgaard and Lafosse (29) described and recommended section of the inner branch of the terminal tendon of the flexor metatarsi. They believed that the lameness could be cured by removing the tension of the tendon and the pressure of the latter on the joint. As Möller has pointed out, however, the only result of the operation was to promote anchylosis of the small joints of the hock by the inflammation which followed it.

The opening of the bursa of the inner branch of the flexor metatarsi, which was first recommended by Dieckerhoff (24), has the same result. The operation is very simple, and for its performance one requires only a broad scalpel with a convex edge. Quiet horses may be operated upon in the standing posture, but restless and vicious animals must be cast. A twitch having been applied, the horse is placed with the diseased side next the wall, and the sound leg is held up as in shoeing. The scalpel, which is held between the thumb and the fore and middle fingers, " is quickly introduced through the skin into the lower part of the bursa, and instantly withdrawn." The hæmorrhage is slight, but the operator must guard against wounding the saphena vein. The opening of the bursa can easily be controlled by means of a sound, and the after-treatment must be conducted according to the rules of surgery. Cicatrisation takes place in about three weeks, and after other two weeks the horse may be cautiously put to work again. Instead of a scalpel one may use the hot iron to open the bursa, but Dieckerhoff prefers the former.

Another, and a very old, spavin operation is periosteotomy. This is said to have been first performed by the English veterinary surgeon Moulden (24, p. 226), and as early as 1849 it was minutely described by Hintermayer. The usefulness of this operation has been proclaimed especially by Peters (43) and Möller (29). Its object is to accelerate the occurrence of synostosis by stimulating proliferation on the part of the periosteum in the neighbourhood of the joint. According to Peters and Möller, it is performed as follows. The horse is cast with the diseased limb undermost; the affected hock is pulled forwards from under the sound leg, and the skin on the inner surface of the hock is thoroughly disinfected according to the rules of surgery. At the lower limit of the hock a transverse incision about 1 cm. in length is then made through the skin and fascia with a convex scalpel, and from this opening the skin in the form of a V is detached from the subjacent tissue by means of curved scissors. Into this pocket of skin a particularly strong and

FIG. 13.

Spavin knive (moderately curved). ¼ natural size.

P

more or less curved flat scalpel (Fig. 13) is introduced as far as the handle, the edge being directed backwards during insertion of the scalpel into the anterior pocket, and forwards during insertion into the posterior pocket. By this means wounding of the vena saphena or of the skin is avoided. The cutting edge is then turned towards the joint, and with the help of the left hand the instrument is pressed in on the bones of the joint with a rocking movement. As Möller has convinced himself on experimental horses, the inner tendinous division of the flexor metatarsi and the periosteum on the cuneiform bones is hereby severed with a loud grating noise, and not rarely the inner long lateral ligament is also cut. Finally the blood is pressed out of the pocket of skin, and an antiseptic bandage is applied. After the operation the horse must stand for four to six weeks. At first a considerable bony swelling forms at the seat of operation, but this afterwards disappears.

Klemm (42), on the ground of his observation that spavin is frequently caused by too low heels and a long toe, has recommended subcutaneous section of the tendinous part of the flexor metatarsi and of a part of the muscular division of the same muscle towards the middle of the tibia. He alleges that in Dieckerhoff's operation it is not the opening of the bursa, but the section of the inner division of the tendon of the flexor metatarsi which takes place at the same time, that is beneficial. Klemm gives the following directions regarding his operation : " The horse having been cast with the affected limb upwards, an incision is made through the skin on the outer side of the tendinous part of the flexor metatarsi, which may be distinctly felt as a cord under the skin, and a disinfected tenotomy knife is introduced. It is advantageous to cut through only a part of the muscular division of the flexor metatarsi along with the tendinous part, for the horse can then more quickly be put to work. After withdrawal of the tenotomy knife the wound may be closed with a pin. After eight to ten days the horse may be put to light work, and after four weeks he may do ordinary work without injury." Klemm's operation has not found favour, and Möller especially has not found it of any use.

Neurectomy of the tibial nerve, which was carried out long ago by Spooner and Stanley (49), has been repeatedly tried in spavin, but as yet without any good result. Almost all veterinary authors are agreed that section of the tibial or peroneal nerves, or of the inner or long cutaneous nerves, has no influence on spavin lameness.

In all methods of treatment great care must be bestowed on the shoeing. If the condition of the hoof permits, the toe ought to be shortened, and a long wide shoe with toe - piece and calkins, or thickened at the heels, applied

None of the before-described methods of treatment has obtained general recognition. Successes and failures have been observed and recorded in connection with each of them. Without doubt firing has the greatest reputation. My own experience warrants me in recommending perforating firing as the best method of treatment. Following Fröhner's method, I perform the operation as follows : The hair is carefully clipped off the inner surface of the hock, and the skin is thoroughly disinfected and neatly dried. While the horse is standing a twitch is applied to the nose, and a fore foot is held

up. With a pointed needle-shaped iron, of the form shown in the annexed figure, I then fire at one or two points directly into the spavin enlargement. The firing iron carries on a stout head of 5 cm. long and about 1½ cm. thick a sharply defined pointed piece 1½-2½ cm. long, about 3 mm. thick at its base, and gradually tapering to its extremity. If the spavin prominence is large I fire percutaneously at a few places round the perforated point. Immediately after firing the entire surface is brushed over with iodoform-collodion. During the next three days the horse is kept in the stall with his head tied up, while the hock joint, the degree of weight put on the limb, and the internal temperature are carefully observed. Attention must also always be given to providing clean straw. The animal must remain in the stable for at least four weeks. During the first days a diffuse swelling forms at the seat of firing, but this soon declines. A slight elevation of temperature is also sometimes observed.

As already indicated, the firing is carried out in the standing posture. By way of preparation, a morphia injection (0·5 gramme) may with advantage be given to very restless animals. As regards the number of points, I consider one or two sufficient as a rule. To fire fifteen to twenty canals, as Hoffman recommends, I consider superfluous. With very large spavins I prefer to fire a few percutaneous points around the perforations.

During the past year I have fired 102 spavined horses in this way, and in many of these a cure was effected. These results are in harmony with the observations of Fröhner, who has also obtained very favourable effects from perforating firing.

Firing directly into the spavin is no new operation, as it was known as early as last century. Since then it has been repeatedly recommended and practised, but it has always been abandoned after a time, on account of disagreeable accidents observed after the firing. As a matter of fact, the operation is not quite free from danger; but that the risk of septic inflammation of the hock joint associated with it is not very great, and can be prevented, is proved by the fact that for a year past I have frequently employed perforating firing in the Polyclinique. Here the animals immediately after the operation must be taken back a longer or shorter distance, which naturally favours the chances of infection; but in spite of this, out of the 102 cases of disease I have only seen one of septic inflammation of the hock joint, and even that one was not entirely free from doubt. Fourteen days after having been fired this horse showed suddenly an extensive swelling of the entire hock joint and suppuration out of the fired canals. Up till then the animal had put its foot well to the ground, but that was now held up, and the entire hock joint was covered with crusts of dirt and dung, the owner having from the outset neglected my order that the horse was always to be kept provided with clean straw. The *post-mortem* examination showed a purulent inflammation of the tibio-tarsal joint and of the neighbouring tendinous sheaths.

FIG. 14.
Iron for perforating firing.
½ natural size.

These untoward accidents may easily be prevented by taking the precautions already indicated. This has already been shown by Fröhner, who has performed perforating firing on fifty-nine horses without losing one of them.

The frequent deaths which formerly occurred after perforating firing I ascribe to insufficient disinfection, to the use of too heavy a firing iron, and to the seat of operation having been too high up. The needle-shaped iron must not be too thick, in order that the subsequent inflammatory reaction may close the canal and prevent infection. The penetrating part of the iron must also be at least 1¼ cm. long, as otherwise it does not reach the bones, and in order to permit of its being pressed into the tissues it must have sufficient hardness. I have always used iron needles; I have found that copper needles are much too soft, and therefore bend easily. In heating the iron in the fire care must be taken to turn the needle upwards, as otherwise it becomes burnt before the head of the iron is heated. The perforations should be made as nearly as possible over the summit of the exostosis, but care must be taken not to perforate the true hock joint, as that is always dangerous. I have not been able to convince myself that there is much danger in opening the small joints of the hock. In three cases I observed a pronounced purulent arthritis of the lower divisions of the hock joint, but this healed up after ten days. On the contrary, purulent inflammation of the true hock joint or of the tendon sheaths almost always runs an incurable and fatal course. I have always warned the owner of the risk arising from this.

These easily avoided drawbacks of perforating firing are more than counterbalanced by the generally recognised advantages of the operation. In the first place, perforating firing is undoubtedly the most effectual of all methods of treatment. The chronic ostitis rarefaciens of the small tarsal bones cannot be better influenced by any other procedure than by direct firing into the bones, which by means of the subsequent inflammation hastens the onset of a condensing ostitis and an ossifying periostitis, with rapid production of anchylosis between the scaphoid and cuneiform bones or between the latter and the metatarsus. To this is to be added the great advantage that the operation can be carried out on the standing horse, and in a few minutes. On account of these advantages Fröhner (51) specially recommends perforating firing in spavin, and my experience also warrants me in characterising the operation as the best method of treatment for this disease.

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THE SITUATION AND ORDER OF DEVELOPMENT OF THE LESIONS IN BOVINE TUBERCULOSIS.

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AN accurate knowledge of the morbid anatomy of bovine tuberculosis is of importance to all those who in the course of their practice have to deal with the disease in the living animal, and it is absolutely

essential on the part of those who in the capacity of meat inspectors have to adjudicate upon the carcases of slaughtered tuberculous animals. For example, a want of knowledge of the common situation of the earliest lesions of the disease in cattle may lead to error in *post-mortem* diagnosis, the animal being pronounced not tuberculous while the most constant seats of the disease have never been examined. Or again, a want of knowledge of the ordinary sequence of tuberculous lesions in the ox, and of the methods by which the disease is spread within the body, may lead a meat inspector to regard as an example of generalised tuberculosis what is in reality a combination of centres of local disease.

In this article the situation and sequence of the lesion will be discussed, and from these and the results of certain experiments an attempt will be made to trace the way in which the disease usually spreads within the body of the ox.

There are two ways in which one might endeavour to trace the order of development of the lesions and the mode in which the disease spreads within the body. In the first place, one might infect in different ways (inhalation, ingestion, etc.) a series of healthy animals, kill them after varying intervals, and compare the lesions found on *post-mortem* examination. Unfortunately, expense is a barrier to the employment of this method on a large scale, for if the point to be elucidated is the order of development of the lesions in the ox the experiments must be carried out on animals of that species. By this it is not meant that no light on the dissemination of tuberculosis within the body of the ox is derivable from experiments on, say, guinea-pigs or rabbits, but merely that such experiments would not be conclusive.

The second method consists in tabulating the results of *post-mortem* examination of natural cases of tuberculosis in the ox, and in endeavouring to deduce from these the probable order in which the lesions have developed. In doing this it is obviously necessary to include a considerable proportion of cases that are at an early, or at least not at a very advanced, stage of the disease. The annexed table (p. 228) is compiled from the detailed records regarding thirty-seven animals subjected to *post-mortem* examination by the writer during the past twelve months. It is necessary to observe regarding these animals that they were all adult cows, and that none of them presented during life any distinct symptom of tuberculosis. They may therefore be taken as average representatives of the numerous apparently healthy but really tuberculous animals to be found in the majority of herds in this country. It is further necessary to state that the examination extended only to the following parts: The lungs, pleura (parietal and visceral), pericardium, heart (exterior), bronchial and mediastinal lymphatic glands, mesenteric lymphatic glands, peritoneum (parietal and visceral), stomachs and intestines (exterior), spleen and liver and their lymphatic glands, kidneys, udder and supramammary lymphatic glands, tongue, throat, and pharyngeal lymphatic glands. The lungs, liver, spleen, kidneys, and the various groups of lymphatic glands mentioned were submitted to very searching examination, by manipulation and close inspection after incision.

[TABLE.

TABLE.

No.	Organs in which Tuberculous Lesions were found.
1	Bronchial and mediastinal lymphatic glands and left lung.
2	Bronchial and mediastinal glands.
3	Left bronchial gland.
4	Bronchial, mediastinal, and mesenteric glands and both lungs.
5	Bronchial and pharyngeal glands and right lung.
6	Bronchial, mediastinal, and mesenteric glands, right lung, pleura, and glands of liver.
7	Bronchial and mediastinal glands.
8	Pharyngeal and mediastinal glands.
9	Bronchial, mediastinal, mesenteric, and pharyngeal glands.
10	Bronchial, mediastinal, mesenteric, and hepatic glands and right lung.
11	Bronchial and mediastinal glands and right lung.
12	Both lungs and mediastinal gland.
13	Bronchial and mediastinal glands.
14	Bronchial and mediastinal glands.
15	Bronchial and mediastinal glands.
16	Bronchial, mediastinal, and pharyngeal glands and right lung.
17	Bronchial and mediastinal glands and left lung.
18	Left bronchial gland.
19	Bronchial and mesenteric glands and left lung.
20	Bronchial, mediastinal, hepatic, and mesenteric glands and both lungs.
21	Right bronchial gland.
22	Mesenteric glands and both lungs.
23	Bronchial, mediastinal, and mesenteric glands.
24	Mediastinal gland.
25	Bronchial and mediastinal glands.
26	Bronchial and mediastinal glands.
27	Right bronchial gland.
28	Bronchial and mediastinal glands.
29	Bronchial, mediastinal, pharyngeal, hepatic, and mesenteric glands and both lungs.
30	Bronchial, mediastinal, and mesenteric glands and right lung.
31	Bronchial and mediastinal glands.
32	Bronchial and mediastinal glands and right lung.
33	Bronchial and mediastinal glands and both lungs, mesenteric glands, pleura, peritoneum, and left mammary lymphatic gland.
34	Mediastinal gland.
35	Bronchial glands.
36	Bronchial, mediastinal, and splenic glands, liver, and right lung.
37	Bronchial, mediastinal, and mesenteric glands and right lung.

An analysis of the table brings out the following interesting facts. By far the commonest seats of disease in these thirty-seven animals were the bronchial and mediastinal [1] lymphatic glands. In five cases the discoverable lesions were confined to the bronchial glands, in two cases to the mediastinal glands, and in nine cases to the two groups of glands. Thus, in no fewer than sixteen cases, or 43 per cent. of the total, visible lesions were found only in these two groups of glands. Lesions were present in one or other bronchial gland in thirty-two cases, and in the mediastinal in twenty-nine, while there was only one case in which both these groups of glands appeared to be healthy.

[1] The glands here named mediastinal are those (sometimes only a single gland) situated in the mediastinum, between the right and left lungs, behind the heart and a little above the œsophagus.

In point of frequency of invasion the lungs come after the thoracic glands, lesions having been present in these organs in sixteen cases, or 43 per cent. of the total. Next in order of frequency come the mesenteric glands (eleven cases, or 30 per cent.) and the pharyngeal glands (three cases, or 8 per cent.). The pleura was diseased in two cases, and the hepatic lymphatic glands in three, while the peritoneum, the liver, a splenic lymphatic gland, and a supramammary lymphatic gland were each once involved.

It is desirable in the next place to inquire what are the usual seats of the disease in animals killed during the advanced stages of the tuberculosis. By way of answering that question the record of the *post-mortem* examination in six very advanced cases of the disease will be here given in detail.

Case I.

Cow killed when at the point of death from tuberculosis.

Post-mortem Record.—Carcase extremely emaciated. The posterior left quarter of the udder is considerably larger than its fellow of the opposite side and it is much indurated. On section the indurated part is found to be caseous and partly calcified. The lining membrane of the teat and milk sinus of this quarter is normal. The left supramammary gland is considerably enlarged, measuring 3 inches in its greatest diameter. On section it is found to be extensively caseated and partly calcified. The remaining three quarters of the udder appear normal. The right supramammary lymphatic gland is about half the size of the left. On section it shows some tubercles. The right and left precrural glands are normal. The peritoneum on the right anterior half of the abdominal wall is studded with tuberculous nodules up to the size of a pea. Caseous nodules are also present in the great omentum. Some tuberculous growths are also present on the parietal peritoneum of the left side. The mesenteric lymphatic glands appear to be without exception tuberculous, some being as thick as the wrist and 5 or 6 inches in length. On section they are found to be caseous and partly calcified. A tuberculous growth is present at some places on the peritoneum of the first and fourth stomachs. The mucous membrane of the small intestine shows numerous crater-shaped ulcers up to the size of a sixpence, and also more extensive diffuse patches of ulceration. A number of submucous nodules are also present in the wall of the cæcum. The peritoneum covering the spleen shows some tuberculous growths, but the substance of the spleen is normal. The liver is extensively adherent to the diaphragm, and while in position it is partly concealed at its upper and lower edges by large tuberculous growths, which on section are found to be caseous and calcified. The hepatic lymphatic glands are enlarged to the size of a fist; these also are caseous and calcified. Many old tuberculous nodules are embedded in the liver substance towards its posterior surface; some of these are as large as an orange and all of them are more or less caseous and calcified. The left kidney is normal. The right kidney contains in its cortex about a dozen yellow, caseous and calcified, tubercles, each about half the size of a barley grain. In this kidney there is also what appears to be a diffuse tuberculous lesion at one point, involving both the cortex and the medulla. The ovaries are normal. There are some tuberculous growths on the peritoneum of the broad ligaments. When slit open the uterus is found to be tuberculous, the mucous membrane and cotyledons in both horns being more or less affected. The left lung is adherent to the chest wall by the medium of a tuberculous growth, which is at least 1 inch thick on the chest wall. At the lower edge of this lung and near the diaphragm there is a large grape-like mass, and similar masses are growing

on the pleural surface of the diaphragm. The bronchial glands are enlarged to the size of a fist and contain old caseous and calcified nodules. The lymphatic glands in the mediastinum are enormously enlarged, one measuring 7 or 8 inches in length by 4 or 5 inches in thickness. The left lung contains numerous areas of caseating tuberculous broncho-pneumonia. The right lung and pleura are in the same condition as the left. The lining membrane of the pericardium and the heart itself are normal. The mucous membrane of the trachea about the middle of the neck shows a tuberculous ulcer of the size of a split pea, and above this there is a nodule with the mucous membrane still intact over it. On both sides some of the suprasternal lymphatic glands are enlarged to the size of a walnut and caseous on section. An iliac lymphatic gland on the right side contains some calcified tubercles, while the corresponding gland on the other side appears to be normal.

Case II.

Cow killed at an advanced stage of tuberculosis.

Post-mortem Record.—Carcase much emaciated. Udder and supramammary lymphatic glands normal. Peritoneum normal. Many of the mesenteric lymphatic glands are enlarged, and on section are found to be caseous and partly calcified. One of the hepatic lymphatic glands is also tuberculous. The small intestine shows numerous crater-shaped ulcers. On one of the pillars of the rumen there are two crater-shaped ulcers, each about the size of a threepenny piece. The liver is normal save for some evidence of fluke invasion. The left kidney is normal, but the right contains one tuberculous nodule. Spleen normal. On the left half of the chest there are some grapy growths on the parietal pleura. The left lung is extensively consolidated by tuberculous lesions in its anterior part, and there are scattered patches of caseating tuberculous broncho-pneumonia in its posterior part near the base. The right lung also contains scattered areas of tuberculous broncho-pneumonia, and towards its base there is a cavity as large as a goose-egg; this is filled with yellow, caseo-purulent material. On each side the bronchial glands are enlarged to about the size of a turkey egg. The mediastinal lymphatic gland is 5 inches long and 2 inches thick; it is caseous and partly calcified. On both sides a pharyngeal lymphatic gland is enlarged and caseous. On the dorsum of the tongue, near its upper part, there is a crater-shaped tuberculous ulcer about the size of a sixpence (tubercle bacilli afterwards found in it).

Case III.

Cow killed at an advanced stage of tuberculosis.

Post-mortem Record.—Carcase much emaciated. The udder and its lymphatic glands are normal. There are some tuberculous growths in the omentum and on the parietal peritoneum along the lower part of the abdominal wall. Many of the mesenteric glands are enlarged and tuberculous. Kidneys show no tuberculous lesions. The hepatic lymphatic glands are enlarged and caseous. Throughout the liver substance there are numerous large tuberculous areas filled with yellow, soft, custard-like, caseous material; the anterior surface of the organ is covered with tuberculous growths which largely unite it with the diaphragm. The peritoneum covering the spleen carries tuberculous growths, but the substance of the organ is normal. Interior of stomach and intestines not examined. Some grapy tuberculous growths are present on the left pleura, and tuberculous growths unite the apex of the left lung to the mediastinum. Both lungs show very marked interlobular emphysema, and contain numerous areas of caseating tuberculous broncho-pneumonia. In the right lung the greater part of the anterior lobe

and the whole of the lobe immediately behind the heart is a solid mass of tuberculous tissue. On this side both parietal and visceral pleura carry grape-like masses. The bronchial glands are enlarged to the size of a goose egg and are caseous and partly calcified. The mediastinal lymphatic gland measures about 7 inches in length by about 5 inches in thickness, and it also is caseous and calcified. The pericardium is normal. The heart is pale and flabby. On either side of the pharynx there is a lymphatic gland containing some caseous nodules. The popliteal, precrural, prescapular, and iliac lymphatic glands all appear to be normal. At the lower end of the cervical part of the trachea there is a caseous lesion still covered by mucous membrane.

CASE IV.

Cow killed at advanced state of tuberculosis.

Post-mortem Record.—Carcase much emaciated. The right hind quarter of the udder is three times as large as its fellow on the opposite side, and throughout the greater part of its extent it is very dense. A small quantity of whey-like material with flocculi in it can be drawn off from the teat (tubercle bacilli had previously been found in this). The mucous lining of the milk cistern of this quarter is at some places injected, and the gland tissue around it, forming a mass as large as the fist, is markedly cirrhotic, with softened caseous centres scattered through it. The rest of the gland is very firm and appears to be in a condition of fine cirrhosis. Except in respect to this firmness it is not markedly abnormal to the naked eye. The right supramammary gland is about 4 inches in diameter and 2 inches thick, and it is partially adherent to the tissue of the mammary gland; on section it does not show any distinct tubercles or caseation. The opposite supramammary gland is free, but enlarged to three or four times the normal; it shows no gross lesion on section. The parietal peritoneum is normal. On the inner surface of the great omentum there is some early *perlsucht* formation. The mesenteric glands are apparently without exception more or less enlarged, some of them to the size of the fist. On section these are found to be firm and caseated almost throughout. The smaller glands show caseating centres. In some of the glands there is commencing calcification. The small intestine contains very numerous tuberculous ulcers, many distinctly crateri-form. There are also present in the wall of the bowel firm, pea-sized, caseous nodules with intact mucous membrane over them. The large intestine and its lymphatic glands are normal. Spleen normal. Hepatic lymphatic glands enlarged and caseating. Liver in a very advanced condition of fluke cirrhosis, with numerous flukes in the bile ducts. No tuberculous lesions in the liver. In each kidney there is a tuberculous lesion at the base of one of the lobules; this appears to have begun in the medulla, which shows distinct caseation, while the overlying cortex appears to be more or less cirrhotic, without distinct caseation. About two-thirds of each lung is the seat of tuberculous broncho-pneumonia with extensive caseation. The caseous matter is almost everywhere soft, and in the right lung there are some cavities up to the size of a hen's egg. The uninvaded part of each lung adjoins its lower edge. Bronchial and mediastinal glands greatly enlarged and caseating. Caseating centres in the prepectoral glands. Suprasternal, prescapular, precrural, tracheal, and popliteal glands are normal. The costal pleura on the right side shows some commencing *perlsucht* formation. Peri-cardium and heart normal. One of the pharyngeal glands on the left side has a caseating centre. In the middle third of the trachea, submucous in position, and near its superior median line, there is a row of discrete, firm yellow tubercles, about the size of small peas; the mucous membrane over these is quite intact.

Case V.

Fatal case of tuberculosis in a three-year-old heifer.

Post-mortem Record.—Carcase much emaciated. The udder (not in milk) and its lymphatic glands are normal. Yellow caseous tubercles in the omentum, and similar growths on the rumen. The mesenteric, hepatic, and gastric lymphatic glands are without exception enlarged and caseating; most of them partially calcified. In the cortex of the left kidney there are about a dozen caseous, partially calcified, yellow tubercles, the largest the size of a pea; about the same number are present in the right kidney. One half of the liver appears to be made up of tuberculous growth in the form of yellow, caseous, and for the most part calcified, nodules of all sizes, from a hen's egg downwards. There is a similar growth on the abdominal surface of the diaphragm, and also on its thoracic side. The spleen is normal in size, but its capsule carries a number of yellow, caseating, partially calcified nodules, some of which are partially embedded in the substance of the organ. A few pea-sized yellow tubercles are also present in the splenic substance. The lungs are crammed with tuberculous areas, and in both there are large caverns containing soft caseous matter. One about the centre of the left lung has a diameter of about 5 inches. The bronchial and mediastinal glands are enormously enlarged, being thicker than one's wrist; they are caseous and largely calcified. On both sides the pharyngeal glands are enlarged to the size of small hen's eggs and caseous. The stomachs are healthy. A large number of crater-like ulcers are present in the small intestine. On each side, under the peritoneum and external to the pelvic inlet, there is a lymphatic gland (about three times the size of a garden bean) with a few yellow tubercles in it. A few similar tubercles are present in the left precrural gland. The suprasternal, prescapular, axillary, popliteal, and right precrural groups of glands appear normal.

Case VI.

Cow killed at an advanced stage of tuberculosis.

Post-mortem Record.—Animal much emaciated. Left half of udder normal, and left supramammary lymphatic gland normal. Right half of udder three times the size of the left, and gland tissue shows diffuse tuberculous cirrhosis. The supramammary gland on the right side is six times larger than the one on the left. On section it is grey and very juicy, but shows no distinct caseation. The omentum shows early tuberculous growths in the form of greyish pink fleshy material. Spleen normal in size and appearance. The whole of the mesenteric glands are enlarged, some to near the thickness of one's wrist, and on section they are nearly all caseated and some of them calcified. The gastric lymphatic glands are also diseased, but to a less extent than the mesenteric. The hepatic lymphatic glands are about normal in size, but one of them contains a tubercle. The liver is normal in size, and its parenchyma shows no tubercles. The kidneys also are free from tuberculous lesions. There is extensive pleural tuberculosis, with adhesions between the lung and chest wall at some places. Tuberculous growths on anterior surface of diaphragm. Prepectoral, bronchial, and mediastinal glands greatly enlarged, caseous, and partly calcified. In each lung there are very numerous caseous areas of tuberculous broncho-pneumonia varying in size and apparently in age. At several places there are caverns varying in size from a cocoa nut to a hazel nut. On close inspection very minute tubercles can be detected in the spongy lung tissue remaining between the solid caseating lesions. The heart and the lining membrane of the pericardium are normal. The pharyngeal glands are enlarged, some to the size of a small hen's egg; and on the course of the trachea in the neck there are several scattered glands varying in size from a hazel nut to three times that

size. Some of the glands (suprasternal) under the triangularis sterni muscle are enlarged to the size of a garden bean, and show caseous tubercles on section. The popliteal, precrural, brachial, and prescapular glands are small in size and normal on section. The lymphatic gland external to the pelvic inlet on the right side is enlarged, measuring about 3 inches in its greatest diameter; it is juicy on section, like the supramammary gland on the same side, but not visibly caseated. The corresponding gland on the left side is about the size of a pigeon's egg, abnormally firm, but not visibly caseated. The ovaries are normal, but the uterine wall is tuberculous; at some places the wall of the left horn is half an inch thick, and shows visible caseation.
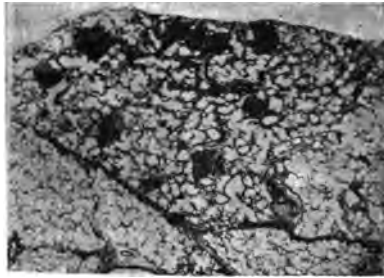
FIG. 1.

Section from spongy lung of Case VI., showing miliary tubercles (×7).

For the most part the lining membrane of the womb is rough, and of a greyish yellow colour. The serous covering of the uterus is normal. The whole course of the intestine, with the exception of the rectum, shows ulcers (numbering hundreds) varying in size from a sixpence downwards, and mostly crater-like, some, however, with the mucous membrane still intact. Corresponding to Peyer's patches there are some ulcerated areas 6 inches in length by 1½ inches in breadth. The fourth stomach shows over a score of crater-like ulcers similar to those of the intestine.

An attempt will now be made to interpret the situation of the lesions in tuberculosis of the ox, in so far as that may be held to indicate the order of development of these lesions, and the methods by which the agents of infection are disseminated in the body.

Lungs and Thoracic Lymphatic Glands.—The parts of the body which are by far the commonest seats of lesions in tuberculosis of the ox are the bronchial and mediastinal lymphatic glands and the lungs, and in a very large number of cases that come under observation in our slaughter-houses the disease appears to have been confined to these parts. It seems impossible to find any other reasonable explanation of these facts than that the common method of infection in the ox species is the inhalation of tubercle bacilli suspended in the atmosphere. When the inhaled bacilli find their way into the pulmonary alveoli they may be carried into the rootlets of the lymphatics (probably by the agency of leucocytes), and thence be transported with the lymph stream until they are arrested in the bronchial or mediastinal glands. Apparently this may happen before the inhaled bacillus has had time to multiply in the alveolus in which it first came to rest, and we may thus explain those cases

in which the lungs are apparently free from disease, and the only discoverable lesions are a few tuberculous nodules in the bronchial or mediastinal lymphatic glands.

In many cases, however, the inhaled bacillus comes to rest in the alveolar cavity or in the alveolar wall, and begins to multiply. The tissues then react to the irritation thus set up, and the evidence of this reaction is the development of a tubercle around the multiplying bacilli. It is conceivable that sometimes the struggle between the bacilli and the animal cells, of which the tubercle is the tangible evidence, may entail the complete destruction of the parasites, in which case the tubercle would cease to grow, shrink, and probably become calcified. As a rule, however, the tubercle bacilli continue to multiply, while the initial tubercle grows and furnishes the seed of new centres of disease, quite detached from the primary one. The growth of the primary lesion is due to the gradual direct invasion of the surrounding lung tissue by the bacilli, and the reaction of the cellular elements of the invaded part to the resulting irritation. In this way a comparatively large tuberculous nodule may come to be developed around the primary point of disease, but almost invariably before that has occurred more or fewer detached new centres are started by bacilli that have escaped from the primary lesion. In the case of the lung there are two different ways in which such new centres are almost invariably set up. In the first place, when the primary lesion has reached a certain size, and especially when it has undergone caseous degeneration or softening, bacilli that have found their way into a bronchial tube involved in the morbid process are apt to be carried in the act of coughing into the bronchi of as yet healthy portions of lung. They may thus set up disease in the bronchial mucous membrane with which they are left in contact, or they may be aspirated into the infundibula or air cells at places even remote from the primary lesion, and there set up fresh centres of disease. In each of these the disease may progress just as it did in the first instance, and thus one can conceive that the settlement of a single living tubercle bacillus might be followed by the development of very numerous areas of caseating tuberculous broncho-pneumonia scattered throughout both lungs. The second way in which bacilli are transported from the primary centre of disease is by way of the lymphatics. It has already been mentioned that inhaled bacilli may probably be carried rapidly out of the lung, from the alveolus into which they have first come to rest, along the lymphatic vessels to the bronchial or mediastinal glands, and this without exciting the formation of any lesion in the lung itself. In many cases, however, the primary lesion is probably in the lung, the disease of the bronchial or mediastinal glands being secondary. Such secondary disease in the latter parts is due to bacilli which at the periphery of the primary lesion have found their way into the lymphatic system of the lung and been carried away in the lymph stream. The transit to the root of the lung, however, is not always direct and rapid. At any part of their course in the lymphatic vessels bacilli may be temporarily brought to a standstill by attacks of the animal cells, and when that occurs a tubercle is developed. Thus, around the primary lesion there may be formed detached lesions along the course of the lymphatic vessels. If the animal survives these may attain such a size as to obscure their mode of origin, and

from them in turn bacilli may be transported either by way of the bronchi or the lymphatic vessels.

By the processes just sketched, viz., primary inhalation of bacilli, continuous direct invasion of the lung tissue from the primary centre, and transport of bacilli by way of the bronchial tubes and lymphatic vessels, one can generally account quite satisfactorily for all the lesions found in the lung. An exception, however, must be made of those cases (exemplified in Case VI., p. 232) in which numerous small or miliary tubercles are found distributed almost uniformly throughout both lungs. The genesis of this comparatively rare lesion will be considered later on.

The Pleura.—It is a matter of common experience that extensive tuberculous disease of the lungs and bronchial and mediastinal glands of the ox is frequently associated with tuberculous pleurisy, and the formation of so-called "grapes" on the serous membrane. Most frequently the pleura becomes involved by direct extension from a lesion in the lung tissue or from a tuberculous bronchial or mediastinal gland, and when once a spot of tuberculous pleurisy is started it may be spread laterally by the lymphatic vessels of the membrane, or by the mechanical dispersion of tubercle bacilli on its surface during the respiratory movements. In this way the visceral layer may infect the parietal, and *vice versa*, and eventually almost the entire surface of the pleura may become involved. When that is the case the lymphatic glands at the entrance to the chest (prepectoral group), and those under the triangularis sterni muscle (suprasternal group), are apt to become infected through the connection of their lymphatic vessels with the pleura, and the disease is also apt to spread through the lymphatic vessels of the diaphragm from the pleura to the peritoneum.

Pericardium and Heart—Tuberculous disease of the heart is very rare. When the heart itself is affected there has been a preceding tuberculous pericarditis, and the myocardium is more or less deeply invaded from without inwards. Tuberculous pericarditis is always secondary to disease of the lungs, pleura, or bronchial glands.

In what precedes, the primary point of disease is supposed to have been in the lungs and to have been the result of the inhalation of tubercle bacilli suspended as dust particles in the air. Let us now endeavour to follow the course of the disease when the agents of infection are introduced into the alimentary canal.

Pharyngeal Lymphatic Glands.—The bacilli may gain the alimentary canal of a previously healthy animal in food or water, or in the case of an animal already suffering from pulmonary tuberculosis they may be ejected from the bronchi, through the trachea and larynx, into the pharynx. In either case the results are the same. The majority of the bacilli that find their way into the mouth or pharynx are swallowed, but not rarely, particularly when they are in large numbers, as, for example, when there is abundant expectoration from diseased lungs, some of them find their way into and through the mucous membrane of the fauces or pharynx. Here they rarely excite a lesion at their point of entrance, but are apparently rapidly carried in the lymph stream to one of the lymphatic glands in the neighbourhood of the pharynx. Within the gland they are arrested, and, proceeding to multiply, they set up tuberculous disease of the

gland. Since the tubercle bacillus is a non-motile organism, one cannot ascribe its entrance into the lymphatic vessels of the throat to its own spontaneous movements; and the frequent absence of any trace of disease in the buccal or pharyngeal mucous membrane, or in the tissues intervening between these and a diseased pharyngeal lymphatic gland, makes it impossible to suppose that the bacilli simply *grow* through the mucous membrane until they find themselves in the rootlets of the lymphatic system, for it may safely be asserted that where tubercle bacilli come to rest and multiply they excite the formation of a lesion. Probably leucocytes play an important part in transporting tubercle bacilli that have been left in contact with the surface of the mucous membrane through the latter, and into the rootlets of the lymphatic vessels beneath the epithelial layer.

Stomachs.—As previously stated, the majority of the bacilli that find their way into the mouth or pharynx will naturally be swallowed, and either directly or after rumination they must reach the fourth or true digestive stomach. It very rarely happens that tubercle bacilli excite disease in any of the stomachs of ruminants, but the writer has seen tuberculous ulceration of both the rumen and the abomasum (*see* Cases II. and VI.) In these instances, however, there had been actual cavern formation in the lungs, with, no doubt, deglutition of enormous numbers of bacilli in the expectorate. The comparative immunity of the three anterior compartments is naturally explained by the thickness of their epithelial lining. That, of course, does not hold good for the true digestive stomach, but the escape of that organ may be due to the acidity of its contents, or to the absence of any tendency on the part of leucocytes to carry away such bacilli as come into contact with the mucous membrane. Probably many of the bacilli that reach a healthy stomach are there destroyed by the gastric juice, but both natural and experimental cases show that when bacilli are ingested in considerable numbers the digestive power of the stomach is a very inadequate safeguard to the intestine.

The Intestine.—When tubercle bacilli reach the intestine they may be carried through its mucous membrane and into the rootlets of the lymphatics in its wall, probably, as in the case of the pharynx, by the agency of leucocytes. Once they have thus gained an entrance to the textures of the intestinal wall they may either be promptly brought to a standstill there, in which case they multiply and excite the formation of a tuberculous ulcer of the mucous membrane, or they may be rapidly transported by way of the lacteals to the mesenteric glands. In these their course is invariably arrested for a time at least, and, proceeding to multiply, they set up tuberculous disease of the gland or glands in which they have come to rest. When the primary lesion in the abdomen is intestinal ulceration the ultimate fate of the mesenteric glands is the same, since sooner or later tubercle bacilli are transported to them by way of the lacteals from the periphery of the bowel lesion. The large intestine is less frequently the seat of ulcers than the small, but it also and its lymphatic glands may become infected by bacilli that have penetrated from its lumen.

Mesenteric Glands.—The most common seat of tuberculous disease within the abdomen of the ox is the mesenteric glands. With perhaps some rare exceptions in which the lesions in them are secondary to tuberculous peritonitis, the mesenteric glands owe their infection to

bacilli brought to them from the intestine by way of the lacteals, and such bacilli may either have been taken in with the aliment or expectorated from a previously diseased lung. When lesions of tuberculous broncho-pneumonia and tuberculosis of the mesenteric glands are present in the same animal it is often impossible to say in which of these ways the glands have become infected, but in a small proportion of cases in adult cattle the lesions are confined to one or more mesenteric glands, and there can then be no doubt that the animal owed its infection to bacilli taken in with food or water. In such cases the number of glands affected is usually not very great. Extensive ulceration of the bowel and tuberculosis of a large proportion of the mesenteric glands are generally the result of a process of auto-infection by the deglutition of expectorate from extensively tuberculous lungs.

Peritoneum.—In a considerable proportion of cases of bovine tuber-culosis the peritoneum is the seat of " grapy " growths similar to those referred to in connection with the pleura. This peritoneal tuberculosis is probably invariably lymphatic in its origin, though some authors have ascribed it to hæmatogenous infection, and viewed it as an indication of generalisation of the disease. There does not appear to be any evidence in support of that view, and the results of the experiments hereafter described are opposed to it.

Tuberculous peritonitis is probably always due to infection of the membrane by bacilli that reach it from a tuberculous ulcer of the bowel, a tuberculous mesenteric gland, or a tuberculosis pleurisy, the last two much more frequently than the first. As in the case of the pleura, the disease spreads by way of the lymphatic vessels of the membrane, and also by the dispersion of bacilli on its free surface during the peristaltic movements of the stomachs and intestines. And, just as in extensive tuberculosis of the pleura the suprasternal and prepectoral glands are apt to become diseased, so also certain groups of lymphatic glands appear to become infected from the peritoneum by way of lymphatic channels. In this way the hepatic, splenic, lumbar, iliac, precrural, and supramammary groups may become diseased. This mode of extension of the disease by way of the lymphatics may be termed *centrifugal*, as distinguished from the *centripetal*, in which the infection spreads in the direction of the normal lymph stream—according to the generally accepted view of the lymphatic circulation. Examples of the centri-petal method of dissemination are seen in the secondary infection of the bronchial glands from a primary focus in the lung, or of the mesenteric glands from an ulcerated intestine.

Liver.—The liver may be infected in several different ways. First—and this is probably the most common — it may be infected centrifugally by way of its lymphatics. In this case the bacilli may reach the organ from its own previously diseased lymphatic glands, or from its peritoneal covering. Secondly, hepatic tuberculous lesions may have a hæmatogenous origin, either by way of the portal or the general circulation. In the former case the agents of infection are brought to the liver in the portal blood, in consequence of an irruption of bacilli into a branch of the portal vein involved in a tuberculous lesion, and in the latter they reach the organ in the arterial blood by way of the hepatic artery. This latter method will be further discussed in connection with the question of generali-sation.

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Spleen.—One of the most characteristic features of tuberculosis of the ox is the rarity with which the spleen is attacked. In an immense majority of cases of tuberculosis in the horse or pig the spleen is one of the seats of disease, but that organ is rarely tuberculous in the ox even when lesions are present elsewhere in the abdomen. In those instances in which macroscopic tuberculous lesions are present in the substance of the spleen of the ox (*see* Case V. p. 232) they are as a rule, if not invariably, due to centrifugal lymphatic infection, either the peritoneum covering the organ or one of the splenic lymphatic glands having been previously diseased.

Kidneys.—The kidneys are seldom found to be the seat of disease in tuberculosis of the ox, though they are more frequently affected than the spleen. Probably renal tuberculosis is also as a rule due to centrifugal lymphatic infection, but it may sometimes be hæmatogenous in origin.

Uterus and Ovaries.—The uterus is not infrequently diseased in advanced cases of abdominal tuberculosis in the cow. The disease generally reaches it by centrifugal lymphatic extension from the peritoneum, but it is possible that tuberculous metritis is sometimes a primary disease, the bacilli being introduced during the act of copulation. The ovaries, which are less frequently diseased than the uterus, are also infected from the peritoneum.

Testicle.—The testicle is one of the organs rarely affected in the bull. Information as to the lesions with which it is usually associated is lacking, but probably infection takes place by way of the inguinal canal from a tuberculous peritoneum.

Udder.—In a very considerable proportion of advanced cases of tuberculosis in the cow the udder is affected. As a rule the condition is associated with tuberculous peritonitis or other extensive tuberculous lesions within the abdomen. There is often evidence that the disease has begun at the upper part of one or other quarter, and one or other supramammary gland is sometimes found to contain tuberculous lesions of considerable standing while the udder itself shows only recent lesions or is quite healthy. All these facts are best explained by supposing that the gland is usually invaded in a centrifugal direction by way of its lymphatic vessels. In harmony with this view is also the fact that the lesion generally takes the form of a diffuse interstitial inflammation of the gland tissue, and not a tubercular (in the anatomical sense) or nodular form such as one would expect if the disease were due to the embolic arrest of bacilli in the capillary vessels of the udder. In occasional cases, however, what appear to be the oldest lesions in the udder are found in connection with the milk-cistern and the lower part of the gland, and it is conceivable that in these cases infection may have taken place by way of the teat canal, perhaps through the agency of a milk syphon soiled with tubercle bacilli.

Bones and Joints.—Tuberculosis affecting either the osseous system or the articulations is comparatively rare in cattle. The bones most commonly affected are those of the spine, and the disease is generally associated with extensive lesions in the abdominal and thoracic glands and viscera. Probably it is generally a local tuberculosis due to infection by way of the lymphatic system of vessels.

Brain and Spinal Cord.—Tuberculous lesions are very rarely

observed in connection with the central nervous system in cattle. In the brain the lesion may take the form of a tumour-like caseating nodule in the hemisphere or elsewhere, or scattered tubercles may be found in the pia mater at the base. In probably all these cases the agents of infection have been carried into the cranial cavity by way of the lymphatic vessels from a previous focus of disease in the lymphatic glands near the base of the skull.

Skin.—The writer has seen only one example of cutaneous tuberculosis in the ox. It affected the skin of a fore-leg below the knee, but no history of the other lesions (if any) present in the animal were obtainable. There can be little doubt that cutaneous lesions when they occur are the result of direct local infection.

Muscular System.—It may safely be asserted that this is one of the very rarest seats of tuberculous disease, if exception is made of those cases in which the muscular tissue in the neighbourhood of a tuberculous bone or joint has become involved by the direct extension of the disease.

Generalisation of Tuberculosis.—At the present day probably everyone is prepared to admit that tuberculosis is for a certain indefinite period after infection a local disease, that is to say, the agents of infection as well as the lesions which they induce are for a time confined to an area in the neighbourhood of the place where the bacilli first obtained an entrance into the tissues. The tubercle bacillus is a slowly growing organism, and one cannot conceive any way in which the entire animal body could immediately become contaminated by the few organisms that may somewhere have obtained an entrance into one of the tissues or organs. This would be true even if the animal tissues and liquids behaved towards the growing bacilli as an inert mass of nutrient material, but, as every one knows, the living tissues *react* to the invading organism, and this reaction impedes the rate of multiplication of the bacilli, and hampers them in their onward march. As has already been mentioned, the bacilli may be moved along or dispersed throughout the body in various ways, such as along the air passages in coughing, by the activity of ciliated epithelium, along the surface of the pleura during the respiratory movements, and along the surface of the peritoneum during the peristaltic movements of the stomach and intestines; but the lymphatic vessels furnish the principal paths by which the bacilli are spread in the solid tissues and organs. With great constancy in susceptible animals more or fewer bacilli escape from the initial lesion into the surrounding lymphatic vessels, and they may then be rapidly transported in the lymph stream. Fortunately, however, they are arrested in the first lymphatic gland on the course of the vessel to which they have obtained access. In the ordinary course the gland itself becomes tuberculous, and after a time bacilli may escape from its efferent lymphatics and again be carried along in the lymph stream. If still another gland is interposed between that lymphatic vessel and the thoracic duct or right lymphatic duct, then a further check in the onward march of the bacilli occurs. Figuratively speaking, the lymphatic vessels are thus the main roads by which the bacilli invade the body, and the various groups of lymphatic glands are so many fortresses which must be reduced by the invaders.

When the last of these has been taken by assault the road is clear, the bacilli reach one of the two great lymphatic trunks, they are thus carried into the venous system at the root of the neck, and they then become dispersed with the circulating blood.

When that has happened the disease is generalised, and until that has happened it is localised. It is obvious that a combination of areas of localised tuberculosis may amount to a very extensive invasion of the body, but without the intervention of the blood current the bacilli cannot be carried into all the organs and tissues. It is necessary to insist upon this point, for the term "general" or "generalised" is sometimes illegitimately applied to cases of tuberculosis in which the disease is merely wide spread in consequence of the body having been invaded simultaneously by way of the lymphatics from several different starting-points, such as the lung, the throat, and the intestine.

The distinction between localised and generalised tuberculosis is one of considerable importance from the point of view of meat inspection, for while there is room for difference of opinion as to the degree of localised tuberculosis compatible with the safe consumption of the apparently healthy parts of the animal, it is obvious that when the disease has become generalised no portion of the carcase is fit for human food. It is therefore important to know how a case of generalised tuberculosis may be recognised when it presents itself in a slaughter-house.

A certain time after the act of generalisation is required for the development of such lesions as may be determined by the bacilli thus dispersed throughout the body, and until such a time has elapsed there will be nothing to indicate what has happened. After the lapse of two or three weeks, however, the lesions excited by the bacilli that have been arrested from the blood will have attained macroscopic dimensions, and the nature of the case will then be apparent on *post-mortem* examination.

In what organs of the body are these lesions to be looked for, and how is a case of generalised tuberculosis to be distinguished from one of widespread localised disease?

Ostertag, in his excellent work on meat inspection,[1] says, with reference to the first of these questions: "General tuberculosis manifests itself in two principal forms. A slight infection of the blood leads to the formation of scattered nodules in various organs, a greater infection to the eruption of innumerable tubercles in most of the organs. In the first case the tubercles generally grow to large nodules or caseous centres by a process of peripheral extension, inasmuch as in this condition the infection of the blood is hardly manifested by any symptom, and therefore does not by itself call for the animal's slaughter (chronic general tuberculosis). In the second case, on the contrary, the tubercles are often met with at a stage when they are but little altered, because as a rule this form of generalisation gives occasion for slaughter (acute miliary tuberculosis). When slight irruptions of tubercle bacilli into the blood are followed by the entrance of enormous numbers, one finds a mixture of these two processes."

"The hæmatogenous lesions, which are originated through the

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[1] "Handbuch der Fleischbeschau." 1st edition, p. 392.

medium of the blood stream, are termed embolic. . . . All the organs which do not immediately communicate with the outer world are the seat of embolic tubercles only."[1]

The same author discriminates between cases of local and those of generalised tuberculosis as follows [2]:—

"The tuberculosis is to be termed local—

"1. When any one organ and its lymphatic glands are attacked, as, for example, the intestine and the mesenteric glands.

"2. In certain cases when two organs are primarily diseased, for example, the tonsils and intestine, with the submaxillary and mesenteric glands; or when along with intestinal tuberculosis only the liver shows embolic lesions.

"3. In disease of the lungs and intestine, when there are no embolic pulmonary lesions, but only tuberculous broncho-pneumonia.

"On the other hand, the tuberculosis is generalised—

"1. When, in addition to a primary seat of disease, widespread embolic lesions (acute miliary tuberculosis) are found in only one organ, as, for example, miliary tuberculosis of the lungs along with tuberculous lesions in the intestine and its lymphatic glands.

"2. When, in addition to a primary lesion, circumscribed embolic lesions (chronic generalised tuberculosis) are found in at least two organs. For example, intestinal tuberculosis combined with lesions in the lung and liver, lung and spleen, or kidneys, etc."

The same author (p. 408) observes that although in most cases of generalisation the muscular tissue is free from tuberculous lesions, other parts of the carcase, such as the lymphatic vessels, the bones, and the lymphatic glands, may be affected. "From a sanitary point of view, in such cases the flesh of tuberculous animals is to be regarded in the same light as tuberculous organs. For the detection of such alterations in the flesh we possess an excellent criterion in the lymphatic glands — the prescapular, axillary, lumbar, precrural, and iliac, as well as those under the vertebral column and those lying on the sternum. When lesions are present in the flesh these lymphatic glands are also affected. In local tuberculosis, on the contrary, these glands are intact, with the exception of the lumbar group, which may be affected in local peritoneal tuberculosis. As a rule, however, it is easy to determine whether the affection is local or general from the absence or presence of embolic lesions in the organs which can only be reached through the blood stream. . . . In judging the flesh of an animal in which generalisation has taken place (tuberculosis of the lungs, liver, spleen, or kidneys) a doubt could exist only in those instances in which the tubercles in the parenchyma are very small. It might then happen that the ordinary macroscopic examination would not suffice for the detection of such small lesions in the lymphatic glands of the carcase, and the examination of these is, for the reasons already mentioned, of the utmost importance. It must be observed, however, that tubercles grow much more quickly and become visible sooner in lymphatic glands than in the parenchyma of the organs. For example, along with lesions not quite so large as a hemp-seed in the spleen one finds as a result of the blood infection quite notable nodules, much larger than a hemp-seed, in the prescapular glands. In order, however, to

[1] Loc. cit., p. 388.　　　　　[2] Loc. cit., p. 394.

be quite safe, it is advisable when embolic tubercles of the size of a hemp-seed are present in the spleen or kidneys not to consider macroscopic examination of the lymphatic glands lying in the muscle as sufficient, but to treat the carcase of the animal as doubtful, and not safe for consumption."

The following three paragraphs relating to this subject are taken from M. Nocard's work, *Les Tuberculoses Animales :*—

"The spleen is rarely the seat of tuberculous lesions. In cases of recent generalisation its tissue is crammed with an infinite number of small greyish granulations, but ordinarily the lesions which one finds are very few in number, relatively large in size, irregular on their surface, calcified, and provided with a fibrous resistant envelope" (p. 26).

"It is clear that in cases of generalisation (acute miliary tuberculosis), all the organs of the abdomen, the spleen, and the kidneys, as well as the liver, are, like the lung, literally crammed with an infinite number of grey transparent granulations hardly as large as a millet seed. These numerous lesions all arrive at the same degree of evolution, proving that generalisation has been effected at the same moment by way of the blood " (p. 30).

"In cases of generalisation the blood and all the vascular tissues are virulent, and those tissues which are favourable to the multiplication of the bacilli, in the first line the liver, the spleen, and the bone marrow, become the seat of a considerable number of specific granulations all of the same volume and of the same age. This constitutes what is known in human medicine as acute miliary tuberculosis" (p. 89).

Nocard, in the work mentioned, also quotes Ostertag's opinion, apparently with approval, to the effect that in the ox species the presence of miliary tubercles in the tissue of the spleen is the best sign that tuberculosis has become generalised (p. 88).

Before proceeding to describe certain experiments which have yielded results that are not in harmony with the opinions expressed in these quotations, it may here be observed that several comparatively familiar facts are calculated to raise in one's mind grave doubts as to the correctness of the view—(1) that splenic or renal lesions are of great importance as an indication that generalisation has taken place ; (2) that tuberculous lesions in the spleen, liver, and kidneys are always hæmatogenous in their origin ; and (3) that the condition of the body lymphatic glands is a valuable criterion in distinguishing between local and generalised tuberculosis. One of these familiar facts is that when a guinea-pig becomes tuberculous as the result of inoculation at the lower part of the abdominal wall or the thigh, the spleen, in association with the inguinal and precrural glands on the inoculated side, may be found extensively tuberculous, while the lungs are still quite free from lesions. Moreover, even when the disease has advanced so far as to invade the lungs, the liver, and nearly all the lymphatic glands of the trunk and limbs, the kidneys are almost invariably found to be normal.[1] Now, to reconcile these facts with the view that the splenic lesions are embolic or hæmatogenous

[1] See article by Professor Sheridan Delépine in this Journal, Vol. X., p. 153.

in their origin, one must suppose that although the bacilli are carried everywhere with the circulating blood, they are for some time allowed to pass through the lungs, but are promptly arrested in the spleen ; or alternatively, that although the bacilli are simultaneously arrested in these organs, the resulting lesions develop much more slowly in the lungs than in the spleen.

Very similar facts are observable in natural cases of tuberculosis in the horse. In that species the primary lesions are nearly always in the intestine or the mesenteric lymphatic glands, and in the vast majority of cases the spleen is affected at any early stage. In these cases of abdominal tuberculosis the lungs either remain free from lesions till the last, or first become affected many months after the formation of the splenic lesions. Here again we should be driven to conclude that although bacilli were being carried everywhere in the circulating blood, and first of all to the lungs, for some inscrutable reason they were not arrested in these organs, or indeed anywhere save in the spleen.

It is, however, not necessary to resort to any such improbable hypotheses in order to account for the appearance of splenic lesions in cases of abdominal tuberculosis, if one views these lesions as lymphatic in their origin.

The statement that the prescapular, axillary, precrural, and iliac lymphatic glands are the seat of lesions in generalised tuberculosis, and unaffected when the disease is local, is also calculated to give rise to some misgivings. In the first place, there are many cases in which the disease would have to be classed as generalised owing to the presence of lesions in the spleen, liver, or kidneys, but which would have to be regarded as local owing to the absence of lesions from the glands mentioned. In the second place, one fails to understand why any of the lymphatic glands are omitted from the groups said to be involved as the result of generalisation by means of the blood stream. On the assumption that the lesions found in the groups mentioned by Ostertag are hæmatogenous in their origin, the almost constant exemption of certain other groups appears utterly inexplicable. It becomes plain, however, when one ascribes the presence of tubercles in the prescapular,[1] axillary, and suprasternal glands to a centrifugal lymphatic infection from extensive lesions in the thoracic cavity, and disease of the precrural and iliac groups to a similar method of infection from a pre-existing abdominal tuberculosis. Under the same supposition the comparative immunity of the popliteal glands follows naturally, owing to their remoteness from the common seats of primary tuberculosis.

The foregoing by no means exhausts all the difficulties in the way of accepting the presence of lesions in one or several of the groups of lymphatic glands of the body (apart from those directly related to the thoracic and abdominal viscera) as evidence of generalisation. It rarely happens that the groups enumerated by Ostertag are all symmetrically diseased, as one would expect them to be if they were infected by way of the blood stream. One frequently finds that several groups of glands on one side are involved while those on the other side are free from disease, and it is quite the rule that when

[1] It ought to be noted also that probably this group of glands may be infected in a centripetal direction, by way of the lymphatics, from a previously diseased pharyngeal gland.

several groups are affected the disease is obviously of a different standing in the various groups.

This, however, appears to be pre-eminently a case in which the divergence of opinion ought to be put to the test of experiment. There is no difficulty, save that of expense, in generalising tuberculosis experimentally in animals of the ox species. All that is necessary is to introduce into one of the veins of the animal a sufficient number of living virulent tubercle bacilli. These will be carried to the right heart, just as they are supposed to be when the disease becomes generalised naturally. They will then be transported to the lungs, and such of them as pass through the capillaries of these organs will be distributed throughout the whole body with the arterial blood. After a sufficient interval the animal may be killed, and the distribution of the tuberculous lesions noted. The following is a record of three experiments of that nature.

EXPERIMENT I.

A piece of horse's mesenteric gland, previously ascertained by microscopic examination to be very rich in tubercle bacilli, was rubbed up in a sterile mortar with a quantity of bouillon; a cover-glass preparation made from the resulting mixture showed immense numbers of tubercle bacilli in every field of the microscope. After subsidence of the coarser particles, three cubic centimetres of the liquid were injected into the left jugular of a cow. This experiment was performed on 18th May 1897.

The cow was killed on 10th June when unable to rise, and the following are the notes of the *post-mortem* examination :—

Carcase very emaciated. Blood not noticeably anæmic. Udder and its lymphatic glands normal. Parietal and visceral peritoneum normal. Peri-
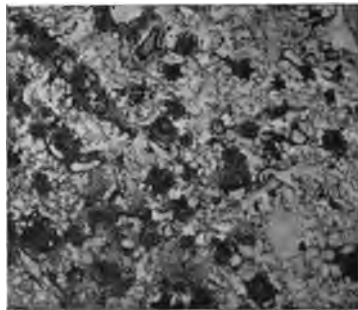
FIG. 2.
Section of lung from cow of Experiment I. showing miliary tubercles (× 7).

toneal cavity contains a small quantity of clear, serous fluid. Mesenteric glands normal. Spleen normal in size; weighs 13 oz. Pulp normal in appearance; Malpighian bodies distinctly visible. Kidneys normal in size; together they weigh 2 lb. 4 oz. Normal in appearance on section. Hepatic lymphatic glands normal. Liver shows slight cirrhosis of the large bile ducts, which contain a few flukes. The organ weighs 14 lb. Adjoining its lower edge there are four spots of localised fatty infiltration, the largest somewhat larger than a garden bean. There is a somewhat smaller spot nearer the gall-bladder. The lobulation is very indistinct, but no tubercles are visible in the organ on section. Right bronchial glands enlarged; the largest is 4

inches long and as thick as two fingers. A turbid juice exudes from the cut surface. The cortical part of the gland is of a dullish white colour, and the medulla is grey. No trace of caseation. Another bronchial gland of this group is as big as a walnut, and still another is half this size; same appearance on section. The left bronchial glands constitute a firm mass as big as a goose egg, and on section they present a similar appearance. Some of the tracheal glands are enlarged to the size of a walnut, and are juicy on section. Mediastinal gland about 5 inches long, and as thick as three fingers. Both lungs show extensive interlobular and subpleural emphysema. The lung parenchyma is uniformly filled with miliary tubercles; these are so close as to be almost confluent. The tubercles are translucent, and about the size of a mustard seed. At some places the lung tissue is quite solid from their confluence. Heart and pericardium normal. Precrural, iliac, and pelvic lymphatic glands normal, as are also those of the large intestine and stomach. Prepectoral, submaxillary, pharyngeal, axillary, suprasternal, prescapular and popliteal glands normal.

EXPERIMENT II.

The subject of this experiment was a cow, which also, on the 18th May 1897, received into the left jugular vein 2 cc. of bouillon holding tubercle bacilli in suspension.

The cow was killed on 23rd June. She was then very ill, and her respiration was gasping. The following are the notes of the *post-mortem* examination :—

The carcase is emaciated. The udder and its lymphatic glands are normal. The peritoneum is normal. The mesenteric glands and those of the large intestine are normal. The spleen weighs a little over 1 lb.; it is normal in appearance, and no tubercles are visible in its pulp. There is an adhesion between the second stomach and the diaphragm, and the adjacent lymphatic glands are enlarged, and contain thick yellow pus. The fourth stomach contains a bullet and several pieces of wire, and its mucous membrane is transfixed by a pin. The liver is normal in size, and no tubercles are visible in it. The kidneys are normal in size; together they weigh 2 lb. 13 oz. No tubercles are visible in their substance on section. The ovaries are normal. The right horn of the uterus contains a quantity of chocolate-coloured fluid. The pericardium and heart are normal. The pleura is normal. The bronchial glands are enlarged to about the size of one's fist. The mediastinal gland is 6 inches in length by 3 in diameter. The cortex in all these glands is beset with opaque tubercles. Throughout both lungs the lobules are almost everywhere partially disassociated by interlobular emphysema; there is also considerable subpleural emphysema. In both lungs the parenchyma is uniformly filled with grey opaque tubercles. One of the pharyngeal glands on the left side is enlarged to the size of a hen's egg, and on section it shows numerous opaque tubercles, the intervening gland substance being congested. On the same side the prescapular gland, although normal in size, shows a few distinct tubercles in the cortex when cut. The axillary and submaxillary glands on the same side are normal. The prescapular gland on the right side is a little larger than the one on the left, but it shows no visible tubercles. The submaxillary, axillary, and pharyngeal glands on the right side are normal. The following groups of glands are normal on both sides—prepectoral, iliac, sublumbar, popliteal and suprasternal.

EXPERIMENT III.

A small quantity of material obtained by scraping the cut surface of a tuberculous lesion in the lung of a cow was shaken up with 20 cc. of sterile bouillon, and after the coarser particles had settled to the bottom of the tube 5 cc. of the supernatant turbid liquid was sucked into a hypodermic syringe

and injected into the left jugular of a two-year-old steer. It had previously been ascertained by microscopic examination that the tuberculous lesion in the cow's lung was unusually rich in tubercle bacilli. The operation was performed on the 8th February 1898.

The steer was killed on the 17th March, when the respiration was very hurried and almost panting (about 60 per minute).[1] The following are the notes of the *post-mortem* examination.

Carcase fairly well nourished (the condition was very good on the 8th February). Peritoneum normal. Stomach and intestines and their lymphatic glands normal (interior of intestine not examined). The spleen is quite normal in size (weighs 1¾ lbs.), and its pulp is normal in appearance. The kidneys together weigh 2¼ lbs. They appear perfectly normal save for the presence in the left one of a small pyramidal pale greyish-pink area (⅛ of an inch at its widest part) in the cortex of one of the lobules. (This was subsequently examined microscopically and found to be histologically unaltered. The pallor was probably due to a deficiency of blood in its vessels.) The liver is normal in size (weighs 13 lbs.), and its parenchyma is quite normal in appearance. Pancreas normal. Between the outer surface of the left lung and the chest wall there is at one point a small firm fibrous adhesion. No fluid in the pleural cavities. Heart and pericardium normal. The bronchial and mediastinal glands are all enlarged, varying in size from a pigeon's to a hen's egg. On section the gland tissue is found to be of a dark red colour, and the cortical part of each is filled with yellow tubercles. At many places these are so close as to be confluent. The medulla of the glands shows no tubercles. When removed from the chest the lungs remain abnormally inflated, and the lobulation is unusually distinct, apparently from congestion and swelling of the interlobular tissue. When the lung tissue is grasped it feels as if its parenchyma were filled with fine sand. On section the lung tissue is found to be everywhere examined with discrete pin-head tubercles, mostly somewhat translucent in appearance. In each lung the tubercles are rather more numerous towards its centre than near the apex and edges. For the most part the lung tissue between the tubercles is spongy, but over a considerable area towards the middle of each lung the lobular tissue is solid, pink, and fleshy in character. The throat and its lymphatic glands are normal. The right prescapular lymphatic gland is one-half larger than the left; it is anæmic and œdematous in appearance, but no tubercles are visible in it. The following groups of glands are quite normal in appearance :—precrural, popliteal, prepectoral, axillary, prescapular, suprasternal, submaxillary, iliac, and lumbar.

It will be observed that, putting aside certain accidental alterations in no way connected with the experiment, there is a very striking uniformity in the distribution of the lesions in these three cases. In each case the lungs were everywhere filled with miliary tubercles, and in none of them could tubercles be detected by macroscopic examination in the liver, spleen, or kidneys. In each case the bronchial and mediastinal lymphatic glands were notably diseased, and, except in Experiment II. the other lymphatic glands of the body appeared to be free from tubercles. The lesions in the bronchial and mediastinal lymphatic glands were unquestionably secondary in character, and due to the transport of bacilli from the lung tissue to the glands by way of the lymphatic vessels. It is highly probable that the tubercles which were present in the pharyngeal and prescapular gland in Case II. ante-dated the experiment, and were the result of natural infection.

[1] The clinical symptoms developed in these cases of generalised tuberculosis were very interesting, but the account of them must be reserved for a future occasion.

In support of that supposition it may be mentioned that the cow in question had been tested with tuberculin on the 17th of May (the day before the experiment was begun) and reacted as follows :—

Temperature at time of infection 101·7°.

    „   „ 6th hour 102°.
    „   „ 9th „   101·6°.
    „   „ 12th „   103·5°.
    „   „ 16th „   103·6°.
    „   „ 19th „   104·6°.

The cow of Experiment I. could not be properly tested as her temperature on the day preceding the experiment was 103·5°. The steer of Experiment III. was tested prior to the experiment and did not react.

What has been said above regarding the results of the *post-mortem* examination in these three cases refers to the lesions revealed by an unusually searching macroscopic examination. It may be objected, however, that although the liver, spleen, and kidneys in these cases were normal to the naked eye they may nevertheless have contained microscopic tubercles. The objection would be valid against the contention that absolutely no tubercles are found in these organs in cases of generalisation, but it would not be valid when the point in dispute is the relative importance of the various organs in slaughter-house inspection for evidence of generalisation. Here there were lung lesions, clearly attributable to the experiment, which could hardly have been overlooked by anyone, while the spleen, liver, and kidneys might have been passed anywhere for healthy. At the same time, considerable interest attaches to the result of the microscopic examination of the last-mentioned organs, which was as follows.

Experiment I.—The liver was found to contain a small number of tubercles—on an average about one for every seven or eight lobules in the section. These tubercles were all of microscopic size, and occurred as small groups of cells within the lobules, where they occupied the place of four or five rows of liver cells. A few of these contained very distinct giant cells.

The spleen contained considerably fewer tubercles than the liver. On an average only one or two were present in each section, the latter measuring ¾ inch by ½ an inch. The tubercles were about the same size or a little larger than those in the liver. Naturally, owing to the structure of the normal spleen pulp, they were less distinct than those in the liver, and might easily have been overlooked had it not been for the giant cells, one or two of which were present in each tubercle.

No tubercle was found in the kidneys, although twenty different sections of the above-mentioned size were very carefully searched with the microscope.

Experiment II.—Not a single tubercle was found in either liver, spleen, or kidney, although twenty sections of each were examined. This fact must be held to strengthen the opinion already given that the tubercles in this cow's pharyngeal and prescapular lymphatic glands ante-dated the experiment.

Experiment III.—In twenty sections of liver examined, fifteen contained one tubercle each, and the remaining five showed none. The largest of these tubercles had a diameter of ·5 mm., and they all contained well-formed giant cells.

In twenty spleen and kidney sections examined not a single tubercle was discovered.

It thus appears that even if a microscopic examination had been resorted to in these three cases of generalised tuberculosis, the liver would have passed for healthy in one of them, the spleen in two, and the kidneys in all three.

By way of illustrating the importance of the lungs as compared with the other organs in examining for indications of generalisation, a return may here be made to Case VI., p. 232. In that instance the presence of minute miliary tubercles in the still spongy lung between the old tuberculous lesions at once indicated that the case was one of generalised disease. The parenchyma of the liver, spleen, and kidneys showed no tubercles to the naked eye, but microscopic examination revealed a few of minute size in each of these organs.

In face of these results it is impossible to maintain that any importance is to be attached to the absence of lesions from the spleen, liver, or kidneys in judging as to whether a case of tuberculosis in the ox has become generalised or not. There is one organ, and only one,
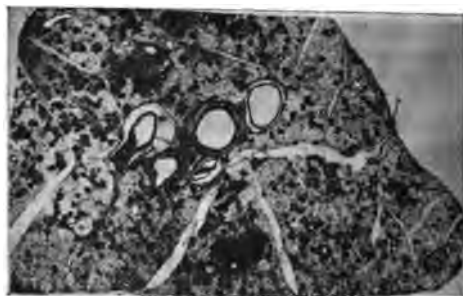
FIG 3.
Miliary tubercles in lung of cow, from a natural case of generalised tuberculosis, (natural size).

whose condition indicates whether the disease is general or local, and that organ is the lung.[1] When tubercles of approximately the same size are scattered throughout the entire spongy lung tissue, as in the accompanying figure (Fig. 3), the case may unhesitatingly be set down as one of generalised tuberculosis. It need hardly be said that the tubercles are not to be looked for in such portions of the lung as had been more or less disorganised by chronic lesions before generalisation took place, and they are sometimes very sparing in number in parts that are in a condition of chronic vesicular emphysema, owing, no doubt, to the comparatively small amount of blood circulating through such portions of lung.

Whenever generalisation takes place by an irruption of tubercle bacilli into a systemic vein, either directly or through one of the two great lymphatic trunks, the bulk of the bacilli appear to be arrested in the pulmonary capillaries, and lead to the development of a crop of tubercles in the lung tissue. Probably in all cases in which the number of bacilli thrown into the blood stream is very great, some

[1] This statement is, of course, subject to the condition that a sufficient time for the development of lesions has elapsed since the act of generalisation.

ass through the pulmonary circulation, and they must then be carried into every organ of the body in proportion to the volume of blood circulating through it.

It is evident, however, from these experiments, as well as from the observed results in natural cases of generalised tuberculosis, that the dissemination of tubercle bacilli with the arterial blood does not determine the formation of tubercles in every part or organ in proportion to the richness of its blood supply. For example, the kidneys, in spite of their large blood supply and the existence of anatomical peculiarities in their circulation favourable to the arrest of particles floating in the blood, are very rarely the seat of tubercles in cases of generalised tuberculosis. The writer has seen many cases of generalised tuberculosis in the horse, with enormous numbers of tubercles in the lung tissue, but he has never met with tubercles in the kidneys in such cases. The same rule, with a few exceptions, appears to hold good in tuberculosis of the ox. The spleen also, as is proved by the experiments previously described, may contain few or no tubercles, although, as shown by those present in the liver, a considerable number of bacilli must have been carried in the arterial blood, and although a far greater quantity of that fluid passes through the spleen than through the liver.

It is sometimes sought to explain these irregularities in the distribution of the lesions in cases of generalised tuberculosis by ascribing them to inequality of circumstances favouring or retarding the multiplication of the bacilli in the substance of the various organs. This, however, seems a far from satisfactory explanation, for while the kidney has comparative immunity from hæmatogenous tuberculous lesions it is now and again the seat of tubercles that apparently owe their origin to an infection by way of the lymphatics. There cannot therefore be anything specially unfavourable to the growth and multiplication of tubercle bacilli within the kidney substance. It is more probable that in some organs few or no tubercle bacilli become arrested from the circulating blood, while in others a considerable proportion are stopped in the capillaries, owing, perhaps, to greater phagocytary activity of their endothelium.

The almost constant differences observed in the distribution of the lesions resulting from the same method of infection in animals of different species are probably due to a different cause. Among such differences may be cited the comparative rarity of renal tuberculous lesions in guinea-pigs and the frequency of the same in rabbits, and the rarity of tuberculous lesions in the spleen in abdominal tuberculosis of the ox and rabbit as contrasted with the comparative frequency of such lesions in the same condition in the guinea-pig, horse, and pig. These differences must be referred to some as yet unexplained dissimilarity in the lymphatic system of vessels in the animals mentioned.

In connection with the subject of generalisation of tuberculosis, one ought to remember the theoretical possibility of an irruption of tubercle bacilli into the blood through the wall of a pulmonary vein. This is a method of generalisation which it would be very difficult to imitate experimentally, and the writer has never seen a case of natural tuberculosis in which the distribution of the lesions suggested that such a thing had occurred.

The results of this inquiry into the subject of generalisation may be summed up as follows :—

1. The never-failing evidence of generalisation is the presence of tubercles of approximately equal size scattered throughout the entire spongy lung tissue.

2. In the absence of the lungs, the other organs, and the carcase generally, do not afford to a naked eye examination reliable evidence as to whether generalisation has taken place or not.

3. In cases of generalised tuberculosis either the kidneys, liver, or spleen, or the whole of these at once, may be free from macroscopic lesions although the lungs contain myriads of embolic tubercles visible to the naked eye.

4. The macroscopic tubercles which are met with in the liver, spleen, kidneys, and lymphatic glands generally, are as a rule due to lymphatic affection, and afford no evidence of generalisation by way of the blood stream.

In conclusion, it must be expressly stated that nothing said in the preceding pages is intended to convey the idea that the apparent absence of macroscopic or microscopic lesions from any organ in cases of generalised tuberculosis is reliable evidence that the organ in question is free from tubercle bacilli and fit for human food. In a future article an account will be given of a series of experiments made to test the infectivity of various parts and organs of the body in cases of generalised tuberculosis.

SOME ABDOMINAL AND UTERINE OPERATIONS IN THE DOG AND CAT.

By F. HOBDAY, F.R.C.V.S., Royal Veterinary College, London.

OPERATIVE TREATMENT FOR HERNIA.

IN the *Journal of Comparative Pathology and Therapeutics* (Vol. X., page 170) a report was made upon twenty-six cases of hernia in the dog and cat which had been submitted to operative treatment. Since then the following nine cases have occurred, making a total of twenty-two inguinal, one scrotal, ten umbilical, and two abdominal. Of the inguinal cases by far the majority were situated on the left side, whilst a fair proportion were double.

The method of operation consists in anæsthetising the animal either with cocaine or some general anæsthetic, cutting down upon the herniated sac under aseptic precautions, and, if possible, returning the contents into the abdomen without opening into it.

When the contents are returned the sac is twisted several times and ligatured, care being taken not to include any of the previously herniated contents. The lower portion is excised and the parts treated antiseptically. If unable to reduce the contents without opening the sac, an incision is made into it and the contents returned with the fingers; or, if thought necessary, as in some uterine hernias, some portion is excised.

The edges of the wound in abdominal cases, or of the ring itself in umbilical and inguinal cases, are lightly scraped and drawn together

with sutures, the parts afterwards being treated antiseptically either with a tent of antiseptic wadding or by the application of orthoform and collodion (1-8) or iodoform and collodion (1-10).

CASE 27.—30th October 1897. Schipperke, female, ten months old, suffering from umbilical hernia. The stitches were removed on the 4th October, the animal making an uninterrupted recovery. The portion of omentum herniated was excised.

CASE 28.—26th November 1897. Scotch terrier, female, seven months old, suffering from umbilical hernia. This case was almost identical with Case 27.

CASE 29.—19th January 1898. Fox terrier, female, four or five years old, with a large inguinal hernia on the left side. The reduction was made without opening the sac, and the patient was discharged from the infirmary with a small external wound on the 25th, making an uneventful recovery.

CASE 30.—3rd May 1898. Spaniel, male, four or five years, with an umbilical hernia about the size of a large walnut. The herniated organ proved to be omentum and was excised, the patient making an uneventful recovery.

CASE 31.—31st May 1898. Toy Yorkshire terrier, female, nine years old, suffering from an inguinal hernia on the left side about as large as a hen's egg. The reduction was made without opening the sac, and the animal was sent home with a small external wound on the 11th of June, making an uneventful recovery.

CASE 32.—13th July 1898. Cat, ten months old, with an umbilical hernia due to protrusion of a portion of omentum. This was excised and the ring sutured, but owing to the protrusion of a still further portion the operation had to be repeated ten days later, the ultimate result being a complete recovery.

CASE 33.—19th July 1898. St. Bernard, female, three months old, with an umbilical hernia about the size of a walnut. The herniated organ proved to be omentum and was excised, the patient making an uneventful recovery, and being sent home five days later. For particulars of this case I am indebted to Mr Browning, M.R.C.V.S., College Tutor.

CASE 34.—17th August 1898. Pug, female, two and a half years, suffering from a large inguinal hernia on the left side. Reduction was unsuccessfully attempted without opening the sac. Upon incising the latter a portion of the body of the uterus and both horns were exposed to view. These were ligatured and excised. The wound healed slowly owing to the mammary gland having been injured, but at the end of three weeks there was only a very small scar to be seen and the patient made a good recovery.

CASE 35.—22nd August 1898. Cat, neutral sex, seven months old, suffering from an umbilical hernia about the size of half a walnut. The stitches were not removed from this, the patient being sent home on the 26th, and making an uninterrupted recovery.

ADENOMA OF THE LIVER.

In January last, by request of Mr Nelder, F.R.C.V.S., of Exeter, I met him in consultation over a Dandie Dinmont bitch, eight years old, supposed by the owner to be suffering from the presence of retained

fœtuses in the uterus.   She had been expected to whelp about the
20th of September 1897, and her abdomen had gradually become
enlarged up to that date.   However, no puppies had . made their
appearance, and the abdomen still continued very much distended.
The bitch did not appear to suffer any inconvenience, having a voracious
appetite, but keeping very thin.

External manipulation revealed the presence of a large nodulated
mass, freely movable inside the abdomen and appearing to occupy by
far the greater portion of it.

An exploratory laparotomy was decided upon, the patient being
secured with hobbles and chloroformed.   The mass proved to be a
large tumour attached to the lower part of one of the lobes of the
liver.   Having passed a ligature around the narrowest part of its
pedicle, the tumour was removed piecemeal in order to avoid making
too large an excision through the abdominal wall.   Unluckily, a lot of
uncontrollable hæmorrhage ensued, the mucous membranes became
blanched, and the patient quietly collapsed just as the last skin suture
was being inserted.

Post-mortem examination revealed all the other organs to be healthy.
The tumour was of a greyish-white colour, with a nodulated exterior,
fairly hard in consistency, and weighed 72 ounces ; in size it was as
large as the head of a child about three or four years of age.   Upon
examination microscopically, Professor M'Fadyean declared it to be
an adenoma.

HYSTERECTOMY DURING PREGNANCY.

This operation, which has several times been reported upon, is
one well worth consideration at times of dystokia, or in cases
where a small female has had the misfortune to become pregnant by
a sire of a large breed.   If performed before the patient has become
weak and in a state of collapse the prospects are very fair, pro-
vided that aseptic precautions are rigidly carried out, the chances of
septic infection being very much less than after Cæsarean section.
The chief disadvantage is that afterwards the animal is useless for
breeding purposes and can only be kept as a pet.   The following
cases were operated upon between July 1897 and July this year :—

CASE I.—5th July 1897.   Cat, three or four years old, which the
owner suspected to be suffering from tumours in the abdominal
cavity.   The animal was ill and somewhat thin, having had a very
irregular appetite for some time.   Suspecting pregnancy, I questioned
the owner very closely, but he was so certain that the animal had
never had any opportunity to meet the male that I decided to perform
an exploratory laparotomy and endeavour to remove the " tumours,"
which could clearly be felt.   These proved to be five fœtuses about
five weeks old, and were removed.   However, the patient refused all
food and died of exhaustion four days later.

Post-mortem revealed nothing to account for death beyond the
empty state of the stomach and intestines, the peritoneal wound not
showing the slightest trace of pus.

CASE 2.—14th February 1898.   Cat, fifteen months old.   The
operation passed off successfully, a few stitches being released on the
17th, and the animal being sent home early in March.   The uterus
contained three fœtuses fully seven weeks old.

CASE 3.—22nd February 1898. Cat, twelve months old. The uterus contained three fœtuses fully eight weeks old. The animal making a good recovery and was sent home early in March.

CASE 4.—23rd February 1898. Cat, two years old. The uterus contained four fœtuses, and, owing to two of the catgut ligatures slipping off, there was, unfortunately, a considerable amount of hæmorrhage. On the 26th a small piece of omentum became herniated and was excised, but on the 28th the animal collapsed and died.

Post-mortem showed the cause of death to be peritonitis.

CASE 5.—June 1898. Toy Yorkshire terrier. A case of fœtal dystokia. The animal had been in labour for some considerable time, and was practically moribund when the operation was performed. An unsuccessful sequel was predicted, and, although the operation itself was a success, the little patient never rallied, death occurring about two hours later.

CASE 6.—13th July 1898. Wire-haired terrier, three years old. This bitch had been lined by a large retriever dog, and, as she had on a previous occasion (when the sire had been a small terrier) had trouble at time of parturition, the probabilities were that she would not in this instance be able to get rid of her pups. The fœtuses were now six weeks old, and the bitch was in very fat condition. The ovaries and the whole of the uterus, which contained four large puppies, were removed. On the 15th a little hernia of omentum and intestine occurred ; these were carefully washed with chinosol lotion and returned, the internal part of abdomen contiguous to the wound being also carefully cleansed. The patient made an uninterrupted recovery, and was sent home in about a fortnight.

The method of operating is as follows :—

The patient is prepared by carefully shaving the hair from the abdominal wall for 2 or 3 inches around the median line between the umbilicus and pelvic brim. A pad of aseptic wadding is then bandaged over this in order to keep the parts clean, and an anæsthetic is administered. The instruments having been boiled, the patient is placed in the dorsal position with the legs well extended, and an incision is made through the skin and subcutaneous fat directly on the median line ; if any hæmorrhage occurs the vessels must carefully be taken up with artery forceps before the abdomen is opened. The peritoneum is punctured and slit up with the aid of a scalpel and director, the fore-finger being inserted and hooked round the uterus ; the latter is brought to the surface and ligatured around the body just below the junction of the two horns and also around each horn just above the ovaries ; two ligatures are put on in each instance, one at the proximal and the other at the distal end, and the incision is made between them. The stumps are returned into the abdomen, the wound in the wall of the latter being carefully cleansed and sutured. For the abdominal wall itself, in each of the cases mentioned above, silkworm gut sutures were used, the muscles and skin being united by sutures of boiled silk. The parts were then thoroughly dried, the wound being hermetically sealed by the application of orthoform and collodion (1-8). Subsequent treatment consisted in the removal of a couple of sutures on the third or fourth day and the application of cleanliness and antiseptics to the wound. In Case 6 a bandage was applied in order to give some support to

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the abdominal wall, but it was not found necessary in any of the others.

In the *Journal of Comparative Pathology and Therapeutics* (Vol. IX., page 157) one case of removal of the uterus and contents in a fœtal dystokia is recorded, the animal dying from septic peritonitis on the fifth day ; and in the same Journal (Vol. X., page 177) two cases are recorded, both of which did well, thus giving a total of nine cases in all, out of which five have made good recoveries. Case 5 was quite hopeless from the commencement, and in Case 4 I attribute the want of success solely to the slipping of the catgut ligatures.

OÖPHORECTOMY AND OVARIOTOMY.

In this Journal (Vol. X., p. 175) there is an account of the method of performing the operation commonly known as spaying in the bitch and cat. Fifteen cases are there recorded, all of them being successful. The following twenty have been operated upon since.

CASE 16.—2nd October 1897. Collie, five months. This animal was turned loose with the other puppies as soon as it had recovered from the effects of the chloroform, and it required no further attention.

CASE 17.—30th October 1897. Manchester terrier, about fifteen months old. There was no pus at all, and the animal made an uninterrupted recovery.

CASES 18 and 19.—15th November 1897, were cats, aged respectively eight and twelve months, and each was sent home a week later.

CASE 20.—14th December 1898. Cat. Everything progressed favourably until the 22nd, when, for some inexplicable reason, symptoms of acute gastritis occurred, death taking place the same evening.

· *Post-mortem* revealed acute gastritis, the stomach and intestines being almost empty. The peritoneal wound had a small piece of omentum adherent to it and enclosed within its edges, but there was no trace of pus in the abdominal cavity.

CASES 21 and 22.—March 1898. Cats, aged respectively three months and two years ; the stitches were not removed, and the animals made uninterrupted recoveries.

CASE 23.—23rd March 1898. Cat, five-and-a-half years, the ovaries alone being removed with blunt scissors and no ligatures applied. This patient went on well until the 25th, when, without any warning, she became suddenly very vicious (showing temper even when looked at) and paralysed in the hind quarters. Death occurred during the evening.

Post-mortem revealed nothing to account for the symptoms, there being not the slightest trace of peritonitis.

CASE 24.—23rd April 1898. Cat, which had been the subject of epileptic fits ever since the arrival of her kittens about a fortnight before. Double oöphorectomy was performed, a kitten being placed with her on the 25th ; she made no objection to suckling it and was discharged from the infirmary on the 3rd of May.

CASE 25.—20th May 1898. Cat, twelve months old. The ovaries only were removed with blunt scissors, no ligatures being applied to the uterus ; an uninterrupted recovery ensued.

CASE 26.—2nd June 1898. Cat, three years, which had had kittens three weeks ago. About ten minutes after the operation was completed the animal suddenly collapsed and all efforts at resuscitation failed.

Post-mortem examination revealed rupture of the liver and the presence of a quantity of blood in the abdomen.

CASE 27.—2nd June 1898. Cat, four years. On the 5th some of the stitches in the abdominal wall gave way, allowing a portion of omentum to escape. A portion of this was cut off and the remainder returned, fresh sutures were re-inserted and the abdominal wound thoroughly washed with chinosol lotion (grs. iv. to ʒvi.). The animal made an uninterrupted recovery, but some of the abdominal sutures have evidently given way again as there is at the present time (September) a perceptible hernia at the seat of operation.

CASE 28.—4th June 1898. Fat Schipperke, ten months old. Uninterrupted recovery, the sutures not being removed at all.

CASES 29, 30, and 31.—June 1898. Cats, aged two years, eight months, and nine months respectively. The older one had had kittens three weeks before. In each case the ovaries only were removed with blunt scissors, and an uninterrupted recovery was made.

CASE 32.—8th June 1898. Very fat pug, three years old, suffering from ulcerating papilloma of the vagina. The papilloma was removed at the same time, and the animal was sent home on the 18th.

CASE 33.—14th June 1898. Cat, about two years, in œstrum, had had kittens about a fortnight before. Boiled silk was used throughout the operation, but the abdominal sutures gave way four days later, allowing the omentum to protrude; a portion of this was excised and the remainder returned. After this recovery was uninterrupted.

CASE 34.—10th July 1898. Pug, two years, suffering from a large ulcerating papilloma of the vagina. Oöphorectomy was performed successfully, but unfortunately the papilloma was excised at the same time. About ten minutes after the completion of the second operation the animal collapsed, and all efforts at resuscitation failed. Although we have on several occasions performed these two operations at the same time I think that it is too severe a test.

Post-mortem revealed no cause to account for death, which must, I think, be attributed to shock.

CASE 35.—13th July 1898. Bull bitch, eighteen months, suffering from ulcerating papilloma of the vagina. After the experience of Case 34 the vaginal tumour was not touched until August, double oöphorectomy being now performed. From this the animal made an uneventful recovery.

For most purposes in which laparotomy is required, the incision in the median line is certainly preferable to that in the flank, for the following reasons :—

1. Hæmorrhage is less, and, in fact, with the median line wound is often so very slight as to be almost imperceptible.

2. If, by some unfortunate circumstance, pus forms in the abdomen, there is a much better chance of obtaining efficient drainage.

3. The wound heals quicker and better.

4. For ovariotomy cases, only one wound is necessary.

5. The scar which is left is not visible unless sought for, and indeed

even then in many cases it can only be found with the greatest difficulty.

As regards sequelæ to the ovarian cases, it has been said that cats and bitches become fat and idle after the ovaries have been removed. This may be so in some instances, but is certainly by no means an invariable rule, and may, I think, often be attributed more to advancing age and want of proper diet and exercise. Several of the animals from which the uterus or ovaries were removed have been under close observation for more than two years, and in the case of dogs they are just as valuable as watch-guards or pets, and in the case of the cats they are just as valuable for catching mice, as they were before.

Summing up these cases of abdominal and uterine surgery, it must be conceded that the abdomen of the dog and cat can be opened without any great dread of the consequences, although, of course, no one should treat carelessly any operation involving laparotomy. With strict attention to asepsis, thanks to the researches of Lister, the surgeon nowadays need not dread interfering with the peritoneal cavity whenever that may be considered absolutely necessary.

EDITORIAL ARTICLE.

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REPORT OF THE ROYAL COMMISSION ON TUBERCULOSIS.

IN our last number we printed the Recommendations of the Royal Commission on Tuberculosis (p. 182), and devoted some pages to the discussion of some of the subjects dealt with in the Report (p. 159). On the present occasion we propose to notice those portions of the Report which treat of milk in relation to human tuberculosis, and of the practical means of eliminating tuberculosis from among our cattle.

It will be recollected that after a careful consideration of all the evidence submitted to them regarding the degree of danger arising to human beings from the fact that tuberculosis is a common disease among cattle slaughtered for the food of man, the members of the Commission came to the conclusion that the risk in this connection had been exaggerated. It cannot be said, however, that the tendency of the Report is to belittle the estimate of the danger attaching to milk which is generally held by those who have paid attention to the subject. In the first place, it was proved to the satisfaction of the Commissioners that tuberculosis prevails to a larger extent among dairy stock than in any other class of animal, and, in the second place, it was brought out in the evidence laid before them that the law has not conferred upon Local Authorities sufficient powers to prevent the sale of milk from tuberculous cows, even when the disease has manifestly attacked the udder.

The Report points out that in both England and Scotland the

Dairies, Cowsheds, and Milkshops Order, 1885, as amended in 1886, is the chief measure whereby Local Authorities can take steps to safeguard the purity of the milk supply. At first sight it might appear that the powers granted in this Order ought to suffice to prevent the sale of tuberculous milk, but, as a matter of fact, the Order, so far as tuberculosis in milch cows is concerned, is a dead letter. It empowers the Local Authority to make regulations—"(a) for the inspection of cattle in dairies; (b) for prescribing and regulating the lighting, ventilation, cleaning, drainage, and water supply of dairies and cowsheds in the occupation of persons following the trade of cowkeepers and dairymen; (c) for prescribing precautions to be taken by purveyors of milk and persons selling milk by retail against infection or contamination." It also directs that "if at any time disease exists among the cattle in a dairy, or cowshed, or other building or place, the milk of a diseased cow therein shall not be mixed with other milk, and shall not be sold or used for human food." Unfortunately, the word "disease" as used in the Order is strictly limited to those diseases included under the Contagious Diseases (Animals) Act of 1878, of which tuberculosis is not one.

The Infectious Disease (Prevention) Act, 1890, also contains some provisions which, one would think, might be put in motion against cases of tuberculosis in milch cows. The Act is one which may be adopted by any Local Authority, and it contains the following clause— "In case the medical officer of health is in possession of evidence that any person in the district is suffering from infectious disease attributable to milk supplied within the district from any dairy situate within or without the district, or that the consumption of milk from such dairy is likely to cause infectious disease to any person residing in the district, such medical officer shall, if authorised in that behalf by order of a justice having jurisdiction in the place where such dairy is situate, have power to inspect such dairy, and, if accompanied by a veterinary inspector or some other properly qualified veterinary surgeon, to inspect the animals therein, and if on such inspection the medical officer of health shall be of opinion that infectious disease is caused from consumption of the milk supplied therefrom, he shall report thereon to the Local Authority, and his report shall be accompanied by any report furnished to him by the said veterinary inspector or veterinary surgeon, and the Local Authority may thereupon give notice to the dairyman to appear before them within such time, not less than twenty-four hours, as may be specified in the notice, to show cause why an order should not be made requiring him not to supply any milk therefrom within the district until such order has been withdrawn by the Local Authority; and if, in the opinion of the Local Authority, he fails to show such cause, then the Local Authority may make such order as aforesaid."

The Report of the Royal Commission states that, although there

are very few districts in which this Act has not been adopted, it has been found quite inapplicable to purposes of tuberculosis or of udder disease generally in the milch cow. The cause of this failure is not very evident, for one would have supposed that at the present day neither medical officers nor veterinary surgeons would have any hesitation in certifying, or any difficulty in convincing the members of a Local Authority, that infectious disease is being spread whenever the milk of a cow with tuberculosis of the udder is being sold for human food.

The Glasgow Police (Amendment) Act, 1890, includes clauses which were intended to remedy these defects in the Dairies, Cowsheds, and Milkshops Order, and the Infectious Disease (Prevention) Act. One of these clauses runs as follows: "Every dairyman or keeper of a byre or cowshed whose milk is sold in the city who, after intimation has been made to him by the Police Commissioners that any cow in his possession, kept for the supply of milk for human consumption, suffers from tuberculosis or any disease which might render the use of such milk for human consumption dangerous or injurious to health, shall retain such cow in his possession, shall, unless the contrary be proved, be presumed to have sold the milk produced by such cow for human consumption, and shall be liable to a penalty not exceeding £5, and such penalty may be sued for and recovered before the sheriff of the county in which such person is domiciled, at the instance of the Procurator Fiscal."

Turning now to the measures necessary to remedy the present highly unsatisfactory state of the law with reference to the sale of milk from tuberculous cows, the Commissioners recall the opinion expressed by Drs Martin and Woodhead, and quoted in the Report of the previous Royal Commission, "That no tuberculous animal of any kind should be allowed to remain in a dairy." While concurring generally with this view, the members of the recent Commission did not feel constrained to recommend that the law should immediately be amended so as to prohibit the sale of milk from a cow affected with tuberculosis in any degree. No evidence was laid before them to prove that the milk is dangerous unless the udder itself is diseased, and, having regard to the extent to which tuberculosis exists among milch cows, they consider that direct action for the elimination of tuberculosis should proceed tentatively.

The directions in which they consider immediate action necessary are: "(1) Systematic inspection of the cows in dairies and cowsheds by the officers of the Local Sanitary Authorities within whose districts the premises are situated; (2) inspection when desired of the cows in any dairy or cowshed, wherever situated, by the authorised officers of Local Authorities within whose districts milk from the premises in question is supplied, on lines somewhat similar to those of Sections 24-27 of the Glasgow Police (Amendment) Act, and of those em-

bodied in the Infectious Disease (Prevention) Act, 1890, and the Public Health (Scotland) Act, 1897; (3) power for a medical officer of health to suspend the supply of milk from any suspected cow for a limited time pending veterinary inspection; (4) power to prohibit the sale of milk from any cow certified by a veterinary surgeon to be suffering from such disease of the udder as in his opinion renders the animal unfit for the supply of milk, or exhibiting clinical symptoms of tuberculosis; (5) the provision of a penalty for supplying milk for sale from any cow having obvious udder disease, without the possession by the owner of a certificate to the effect that such disease is not tubercular."

Last, but not least, among the measures recommended is compulsory notification of every disease of the udder in cows, whether in public or in private dairies.

It is very likely that these recommendations will be considered unnecessarily drastic by farmers and dairymen, but that opinion will not be shared by anyone who is in a position to form a correct estimate of the dangers to which the public are at present exposed owing to the virtual absence of any restrictions on the sale of such a deadly germ as the tubercle bacillus in milk. Indeed, it may be questioned if the recommendations made by the Commissioners embody all the precautions that, having regard to the real nature of the danger, would deserve to be called reasonable.

It is probably true, as suggested in the Report, that the milk is not dangerous unless the udder is itself tuberculous, and if the recommendations above quoted were embodied in the law there would be little cause for complaint so far as regards the sale of milk from cows with recognisably tuberculous udders. Systematic frequent inspection by properly qualified persons, coupled with compulsory notification of udder disease in any form, and power to prohibit the sale of milk from cows certified to be clinically tuberculous, would no doubt go a long way to prevent the sale of milk containing tubercle bacilli, but it must be remembered that in probably every case of mammary tuberculosis there is a period during which the milk is infective although the udder itself is not obviously diseased. No doubt the Commissioners had this fact in their minds when they recommended that Local Authorities should have power to prohibit the sale of milk from any cow certified by a veterinary surgeon to be exhibiting clinical symptoms of tuberculosis, but it is matter for regret that they did not also recommend the compulsory notification of every case of tuberculosis in milch cows manifested by clinical symptoms. In view of the fact that the disease is usually well advanced before any marked symptoms such as emaciation and cough are exhibited, and seeing that in a very considerable proportion of cases of advanced tuberculosis the udder becomes affected, the sale of milk from a cow showing pronounced symptoms of disease is

absolutely indefensible. There is great reason to fear that the time is yet far distant when Local Authorities everywhere will be able to undertake systematic and frequent inspection of all the milch cows in their districts, and in the meantime compulsory notification of symptoms of tuberculosis, with a smart penalty for not reporting, would do much to rid our byres of those "piners" which are a serious source of danger to the milk-consuming public, and a cause of loss to their owner since they inevitably spread the disease to their companions.

Here again the question of compensation comes in. It will be recollected that the members of the Royal Commission were divided in opinion as to the justice or expediency of granting compensation for the carcases of animals condemned on account of tuberculosis in slaughter houses. They were, however, unanimously of opinion that claims for compensation owing to the condemnation of a milch cow affected with disease of the udder or showing clinical symptoms of tuberculosis should not be entertained. They therefore recommend "that when, under the certificate of a veterinary surgeon the sale of milk from a given cow is prohibited, the Local Authority should slaughter the same, and if, on *post-mortem* examination, it appears that the cow was not so affected the Local Authority should pay compensation to the extent of the full value of the cow immediately before slaughter. If on the other hand the animal be found to have been so suffering, the carcase should be sold by the Authority and the owner thereof should receive the proceeds of the sale."

The Report of the Royal Commission says that "all precautions against the communication of tubercular disease to human beings by the consumption of the meat or milk of diseased animals must be regarded as temporary and uncertain palliatives so long as no systematic attempt is made to reduce the prevalence of the disease among the animals themselves." The consideration of the practicable measures of prophylaxis was therefore regarded as the most important part of the inquiry. The Commission took steps to ascertain the methods which have already been put into operation against bovine tuberculosis in foreign countries and the degree of success which the different methods had yielded. The Report characterises as quite impracticable the proposal to stamp out tuberculosis by means of slaughter on the lines followed successfully in the case of such diseases as pleuro-pneumonia and cattle plague. This opinion was partly based on an examination of the results obtained in Belgium, where a scheme of compulsory notification and slaughter, coupled with partial compensation to owners, was initiated by the Government in 1895. After two years experience the scheme had to be abandoned, as "it was found impracticable, and the cost of it intolerable."

But while the Royal Commission thus regard any attempt to stamp

out tuberculosis by the compulsory use of tuberculin and the prompt slaughter of all infected animals as fore-doomed to failure, they view with favour the less radical but more economic plan which is specially associated with the name of Professor Bang. This method has been practised on a considerable scale and with a gratifying degree of success in Denmark. As is well known, the essence of the Danish plan consists in employing tuberculin to ascertain which animals are affected, and in effecting a permanent separation between the healthy and the diseased. The method is voluntary, but the State encourages its adoption by supplying, free of cost, the necessary tuberculin and the services of a veterinary surgeon to carry out the test.

The Royal Commission recommends that similar inducements to gradually get rid of the disease should be offered in this country, and they do not apprehend that there would be any general disinclination on the part of British farmers to avail themselves of such assistance.

The recommendations made by the Royal Commission include several that are intended to bring about an improvement in the hygienic condition of cowsheds. It is recommended that in future all cowsheds should have an impervious floor, a sufficient water supply for flushing, proper drainage, a depôt for the manure at a sufficient distance from the byres, and sufficient light and ventilation. Furthermore, in the case of all cowsheds in populous places a minimum cubic space of 600 cubic feet, and a minimum floor space of 50 feet, for each adult beast is to be provided. These appear to be among the least practicable of the recommendations made in the report. It hardly seems as if the Commissioners had calculated what an enormous sum of money would be required to bring all the cowsheds in the country up to this standard, or taken into consideration the gross injustice that would be inflicted on British agriculture by exacting such a high hygienic standard in the case of home byres, while admitting foreign dairy produce from countries where no such restrictions are imposed on farmers and dairymen.

· And if the Commissioners expected that the suggested, improvements in cow-byres would in any important degree diminish the prevalence of tuberculosis among dairy cows, then many people will not agree with them. The recommendations are calculated to raise in the minds of Local Authorities the idea that if they desire to eliminate tuberculosis from dairy cows the great thing is to insist upon a large cubic space per animal in the cowsheds in their district. What really requires to be brought home to Local Authorities, and also to the owners of dairy cows, is that the most liberal cubic space per animal in cowsheds will not keep a stock free from tuberculosis, and that entire freedom from the disease may be obtained although the buildings from a sanitary point of view may be far from perfect.

Before bringing this article to a close it may not be out of place to

consider what are the probabilities that legislative effect will in the near future be given to all or any of the recommendations made by the recent Royal Commission, and to take stock generally of our present position with regard to bovine tuberculosis. We may regard the disease from two different points of view. In the first place, it is a disease etiologically identical with the one of the same name in human pathology, and it is impossible to deny that in existing circumstances the widespread occurrence of tuberculosis among cattle is to a certain degree a menace to public health. In the second place, and putting this aspect of the disease entirely out of sight, tuberculosis of cattle is a contagious malady which is at the present time detracting from the legitimate profits of agriculture, and to that extent interfering with the national prosperity. In which of these respects the disease is most important it would be difficult to say, but there can hardly be a doubt that the main impetus to inquiry regarding the extent to which it prevails, and the feasible measures for checking or exterminating it, has come from its supposed connection with human tuberculosis. The investigations of the past few years, and the labours of the Royal Commissions which have considered and reported upon the subject, have upon the whole tended to show that the connection between bovine and human tuberculosis is not so intimate or important as was at one time supposed. At least with regard to the consumption of the flesh of tuberculous animals there is a consensus of opinion that the danger is not a pressing one. It is true that the evidence incriminating milk as an etiological factor in human tuberculosis has grown rather than diminished within recent years, but the general body of the public do not appear to have realised that the danger in this connection is a very serious one. This apathy or ignorance of the general public with regard to the sanitary importance of bovine tuberculosis has an important bearing on the prospects of legislation directed against the disease. Any attempt at legislation on very drastic lines would necessarily trench upon many vested interests, and the prospect of the vigorous opposition which would thereby be excited in interested quarters is certain to have a deterrent effect on legislation as long as there is no very general or pressing demand that the control of the disease should be taken in hand by the State.

Then, turning to the importance of tuberculosis from the economic or agricultural point of view, it may be doubted whether there is on the part of farmers and cattle-owners generally any very urgent desire that the Board of Agriculture or Local Authorities should put in force the measures necessary to eradicate the disease. It is true that agriculturists and butchers have frequently expressed great dissatisfaction with the present state of affairs, but when their complaints are analysed it will generally be found that they resolve themselves into a demand that they shall be compensated for the

losses incurred through the condemnation of tuberculous carcases, and that they are by no means anxious for the introduction of such drastic measures as compulsory testing with tuberculin and enforced slaughter of every animal that reacts, even if these were accompanied by moderate compensation.

For these reasons it appears more than probable that efforts to deal with tuberculosis will for some time to come be left to the voluntary initiation of cattle-owners. An exception may perhaps be made of tuberculosis in milch cows, for when the public come to learn of the real magnitude of the danger in this connection, the Government may be compelled to take the disease so far in hand as to legislate against the sale of milk from a cow that has tuberculous disease of the udder or is manifestly in an advanced stage of tuberculosis. It is obvious, however, that restrictions of that character might be in operation for a century without sensibly diminishing the prevalence of tuberculosis among cattle generally.

It may be said that this is a very despondent estimate of the future, and that it sounds like a counsel of passive resignation in face of a state of things fraught with danger to human life and entailing steady losses to agriculturists. Such, however, is not the case, for it does not follow that nothing can be done or will be done because the State refuses to intervene. Even if the recommendation of the Royal Commission that the Government should supply tuberculin and the services of a veterinary surgeon be not adopted, that need not deter the owner of a tuberculous herd from attempting to get rid of the disease. If his losses are so slight that he feels it would not be worth while to buy tuberculin and employ his own veterinary surgeon in order to put a stop to them, the case is hardly one demanding State assistance. On the other hand, if anyone is now experiencing serious loss from tuberculosis among his cattle it may fairly be said that the fault is his own.

It is the duty of veterinary surgeons to make this known, and to persuade the owners of tuberculous herds that it will pay them better to attack the disease now than to wait in the expectation that the State will step in and buy their affected animals.

Reviews.

Veterinary Obstetrics. A Compendium for the use of Students and Practitioners. By W. H. Dalrymple, M.R.C.V.S. New York: W. R.
    Jenkins, 1898.

THIS little book makes no pretension to be an exhaustive work on veterinary
obstetrics, and the author disclaims any desire to see it take the place of the
larger text-books on the same subject. At the same time, we consider it a
useful addition to the student's list of manuals. As far as is possible within
such modest limits (158 pages), Mr Dalrymple describes the anatomy of the
female generative organs, the physiology of pregnancy and parturition, and the
pathological conditions incidental to gestation and delivery of the fœtus.
The language of the work is clear, and there are fifty-one good illustrations.

Friedberger and Fröhner's Veterinary Pathology. Vol. I. Infective Diseases
    of Animals. Translated and edited by M. H. Hayes, F.R.C.V.S.
    Authorised translation. London: W. Thacker & Co., 1898.

FROM the moment of its appearance in German Friedberger and Fröhner's
work has been everywhere recognised as a standard treatise, and indeed it
may unhesitatingly be described as the most comprehensive and scientific
work on veterinary pathology and therapeutics that has yet appeared. We
therefore extend a hearty welcome to the English translation of it. Upon the
whole Mr Hayes has executed his task of translator in a highly creditable
manner, but here and there one detects evidence of haste and want of care
in the translation. For example, we read (p. 339): "The spores of anthrax
are formed only outside of the animal body by the free ends of the bacilli becoming continually elongated." This, of course, is hardly a correct statement, and
it is not an exact rendering of the original. In the same paragraph from which
we have quoted the following curious sentence occurs: "The bacilli are produced from the spores by the spores becoming elongated in the direction of
their long axis towards one side in the form of an oval, cylindrical process, at
one end of which the spore is situated." To endeavour to figure to one's self the
process here described imposes a severe strain on the intellect. How can a
thing become elongated in the direction of its long axis to one side? If it
grows to one side it cannot be elongated in the direction of its long axis.
What is the shape of a body that is at once oval and cylindrical! And how
does the spore elongate as a process at one end of which the spore is situated?
In fairness, however, to Mr Hayes it must be stated that for the most part the
obscurity of this sentence belongs to the original.

In a work which deals almost exhaustively with veterinary pathology and
therapeutics there must necessarily occur many expressions of opinion to
which everyone will not assent, and also some absolute errors. It is the unenviable fate of those who aspire to producing such comprehensive text-books
that they must write regarding many things of which they have had little or
no experience. They have then to rely on the published observations of
others, and in spite of the greatest discrimination some erroneous opinions
and inaccurate descriptions are certain to be repeated. The present text-
book is no exception to this rule, and if time and space did not forbid we might
cite from it many statements that are certainly erroneous, and others that are
at least open to serious doubt. One or two must suffice. On page 293 it is

stated that "Schütz has proved that mallein must not be regarded as a typical reagent for glanders." It need hardly be said that he has done nothing of the kind. All that Schütz has proved in that connection is that some bad preparations of mallein are in use in Germany.

On page 365, under the heading, "Anthrax in Sheep and Goats," we find the following :—"Anthrax in sheep has been confused most frequently with malignant œdema. The affections which Haubner and others described as anthrax, and which were characterised by crackling swellings of the hind limbs, were certainly malignant œdema." Now, that is very far from certain. It is far more probable that the cases referred to were quarter-evil.

From the article entitled "Swine-Fever" one might extract some scores of entirely erroneous statements, the authors having mixed up in inextricable confusion two perfectly distinct diseases, viz., swine-fever and swine-plague. Indeed, we would strongly advise those who purchase the book to seal up the chapter on swine-fever.

But in spite of such inaccuracies as these the work is superior to any other that we possess on the same subject, and it deserves to have a wide sale. It is to be hoped that Mr Hayes will soon be able to issue the second volume necessary to complete the translation.

CLINICAL ARTICLES.

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CONCRETIONS IN THE GUTTURAL POUCHES.

By WILLIAM A. DELLAGANA, M.R.C.V.S., London.

CASES of this nature being somewhat rare in veterinary literature, I venture to describe one which came under my notice. It may be interesting to some of the readers of this Journal.

When stationed at Bangkok, Siam, my attention was one day drawn to an aged Australian mare—a trooper attached to the riding-school. The sergeant in charge complained that the animal was neither able to eat nor drink, and that when she attempted the latter the water returned through the nostrils. I found her standing in her stall in a very distressed condition, forelegs wide apart, head and neck straightened, breathing with difficulty, and quite incapable of lowering her head or even turning it to either side. She had been at work up to within a few days of my seeing her. Upon manipulation in the parotideal region, hard and tense swellings could be felt on either side, which did not yield to moderate pressure or in the least fluctuate, as one would expect of an abscess. There was a slight increase of temperature (101° F.), and an entire absence of cough. The treatment adopted was with a view to relieve pain and at the same time to encourage "pointing," and thus locate a favourable spot for surgical interference. However, some hours after I had seen the mare it was reported to me that she had suddenly dropped down in the stable and expired. A *post-mortem* examination made the following morning revealed lesions of intense inflammatory action. The tongue (root and dorsum), post-nares, pharynx, and larynx were alike

affected. The guttural pouches were literally crammed with concretions and pus, which, on removal, filled an ordinary stable bucket of about three gallons capacity. It size the concretions varied from that of a pea to a hazel nut. The pus was thick and creamy, and not putrid. The lungs were congested and the mucous membranes dark in colour. The immediate cause of death, no doubt, was asphyxia.

The most remarkable features in this case were (*a*) the slight increase in temperature; (*b*) the absence of cough; and (*c*) lastly, but by no means least, the enormous quantity of concretions and pus present in the pouches.

Unfortunately I could not ascertain the history of the case prior to the time when I first saw the animal.

SOME UNCOMMON PARASITES.

By F. HOBDAY, F.R.C.V.S., Royal Veterinary College, London.

FILARIA HEMORRHAGICA (RAILLIET).

In June of last year one of the patients in the Free Clinique was a roan pony, four years old, which had been bought a short time previously out of a Russian drove. It was brought up for advice regarding the presence of a number of lumps in various parts of the body, particularly the shoulders and centre of the back, some of which bled occasionally without any apparent cause. Not suspecting parasites to be the cause, alterative medicine was given internally, and refrigerant lotion applied to the skin; this did not give any permanent success, and although the individual lumps would disappear for a time, after discharging a small stream of blood, there were always fresh ones appearing. Thinking the case to be one of more than ordinary interest, I showed it to M. Montmartin, a veterinary lieutenant in the French army who happened to be on a visit to the College, and he at once diagnosed it as being due to a small subcutaneous nematode of which he had had some experience when buying Hungarian horses for the French cavalry.

With the owner's consent a piece of skin directly over one of the nodules was excised, and, by good fortune, a specimen of the parasite secured. When first seen it appeared like a piece of thin white cotton twisting itself about in all directions; it was seized carefully with a pair of forceps and taken into the pathological laboratory. Upon being placed in water it moved about freely and ejected a large number of ova, this process also being witnessed when put under the microscope. When examined it was found that one extremity was badly lacerated, and that only about three-quarters of an inch of the parasite was present. Professor M'Fadyean proclaimed it to be the Filaria hemorrhagica. On several subsequent occasions we tried to obtain other specimens but always without success. The treatment adopted consisted in the application of iodine ointment with friction; this had the effect of causing the parasite to change its position and so allow the bleeding spot to heal, but fresh sores almost invariably developed in places a short distance away. In September there were

still a few nodules to be felt on each side of the forequarters, but no bleeding sores, and the animal was working regularly; during the winter months they seemed to disappear, but in March of this year the bleeding recommenced just as in the spring of 1897, the iodine ointment being used with temporary benefit as before.

A second case, also in a Russian pony, has recently come under my notice whilst in the country, the symptoms being almost identical with those described above, although the hemorrhagic lumps were not quite so numerous.

Referring to Dr Fleming's translation of Neumann's "Parasites of the Domesticated Animals," I find the statement that this affection is not uncommon in Hungarian horses and horses of the Steppes breed. Dr Fleming also states in an appended note that so far as he is aware the disease has not been witnessed in England except among some Hungarian horses which were purchased for trial by our cavalry some years ago. It is stated that in France it has been observed that the affection if left alone disappears in about three or four years.

PLEROCERCOIDES BAILLETI (RAILLIET).

In March last I received some very curious parasites from Mr Frederick Taylor, M.R.C.V.S., of Birmingham, with the following history.

The patient in whose body they were found was a fox terrier dog, seven years old, born in Malta, and which had been there again two or three times.

On the 9th the animal seemed ill and would not feed, barking continually in a peculiar manner; if left alone it tore up its bed and scattered it about, running all over the house, hiding in corners, and under the beds. The tongue was frequently protruded and of a livid colour. When placed in Messrs Taylor and Burnett's infirmary (12th March) it tore up a mat bed, drank water greedily, but would not eat, and was continually barking. When struck with a whip the animal appeared quite insensible to pain and took no notice whatever of the punishment. In the afternoon when turned loose in the shoeing forge the dog rushed about, barking all the time, and attempted to bite a groom who was near. Having been carefully secured, the throat was examined but without revealing anything to account for the symptoms, and, by the owner's consent, a dose of hydrocyanic acid was administered.

Post-mortem revealed the stomach to be empty with the exception of some straw. The thoracic and abdominal cavities were positively crowded with worms, none of them being found inside the intestinal canal. There were some slight signs of pleurisy and peritonitis, and all the other organs were normal.

From the symptoms shown, rabies was suspected, but inoculation experiments, made at the Board of Agriculture, gave a negative result.

The worms averaged about $1\frac{1}{2}$ inches in length, and they had a peculiar thick anterior extremity gradually tapering off towards the tail. They were exactly like the illustration given in Neumann's " Parasites of the Domesticated Animals," on page 554, and from that they have been identified as belonging to the rare Plerocercoides Bailleti.

A CASE OF VERMINOUS DIARRHŒA IN A BULLOCK, SUCCESSFULLY TREATED WITH LYSOL.

By ROWLAND G. SAUNDERS, M.R.C.V.S., Colonial College, Hollesley Bay.

THE patient, a sixteen-months-old Shorthorn bullock, had been grazing on low lying land (formerly marshy but now drained by dykes), and receiving in addition a liberal allowance of cotton cake. When seen a week previously by the farm superintendent the animal to all appearances was in perfect health and doing well.

I saw him on 6th July, when he had fallen away in condition to an alarming extent. He was "tucked up," with hollow flanks, "hide bound," had a loose and harsh coat, and showed marked weakness of the hind quarters. Pulse 56, small and weak, temperature 102·9°, and respirations 30. He was suffering from a very fœtid diarrhœa, the fæces being green coloured and containing much undigested material. His tail and quarters were bespattered with fæces. He had not lost his appetite, chewed his cud, and had a healthy dew on his nose. Although I was unable to detect worms in the fæces, the symptoms and the fact of the case yielding to the following treatment indicated a verminous origin.

I ordered him to be kept indoors, to be fed on corn and hay, and left instructions for the following drench to be given each day in a quart of starch gruel.

$$
\begin{array}{lll}
\text{R.} & \text{Ol. Terebinth.} & \quad . \quad . \\
& \text{Tinct. Opii} & \quad . \quad . \\
& \text{P. Cretæ prep. āā .} & \quad . \quad \text{ʒij.} \\
& \text{Chlorodyni} & \quad . \quad \text{ʒss.} \\
& \text{Ol. Menth. Pip.} & \quad . \quad \text{mxxx.} \\
& \text{Ol. Lini} \quad . \quad & \quad . \quad \text{ʒvj.} \\
& \text{M. Ft. haust.} & \quad . \quad .
\end{array}
$$

This treatment was persisted in for a week, and on 14th July I saw him again, and found that although he still retained his appetite the diarrhœa was, if anything, worse.

Having heard that Professor M'Fadyean had recently treated some of these cases successfully by administering lysol with the stomach pump, I thought this a good opportunity to try its efficiency but was not sure of the dose recommended by him. On 16th July I administered by means of the stomach pump 3 ounces of lysol in 2 gallons of tepid water. After the first stroke or two of the pump the animal regurgitated a small quantity of ingesta, but afterwards, by pausing for a few seconds between every few strokes, the whole was injected without further trouble.

On 18th July he was decidedly better; the fæces were firmer in consistence, and the fœtid character was entirely absent. I injected another 2 ounces in a gallon and a half of water.

On 20th July a further improvement had taken place, and another 2 ounces were administered. Two days later the fæces had quite returned to a normal consistence and an improvement in condition was appreciable. The lysol was discontinued and a full dose of magnesium sulphate was given. A few days afterwards the animal was turned out again, and has been doing well since then.

DO THE RIGHT AND LEFT PLEURAL SACS OF THE HORSE COMMUNICATE IN PLEURISY?

By HENRY GRAY, M.R.C.V.S., Kensington.

MÖLLER in his "Operative Veterinary Surgery" (Dollar's translation) says on p. 210: "Where both pleural sacs contain fluid, the operation (of tapping) should be performed on the right side, to prevent injuring the heart." Of course, he was referring to the horse when he wrote this. If he means that by tapping the right side the fluid of the left side will also be drawn off then he must have been speaking from imagination and not from actual fact. According to my experience, the pleural sacs of the horse do not communicate, at least after the first stage, in cases of pleurisy. I have tapped four horses this spring, and found that the fluid drawn off from one side differed from that drawn from the other. In two cases the fluid of one side was milky white and fœtid, and that of the other side was amber tinted, clear, and odourless. In the other two cases the fluid on one side was port-wine tinted, and on the other side amber tinted. ·

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ROYAL VETERINARY COLLEGE, LONDON.

LIST OF BURSARIES, MEDALS, HONOUR CERTIFICATES, ETC., 1897-98.

Coleman Prizes.

Silver Medal	C. Radway.
Bronze Medal	H. A. Woodruff.
Certificate of Merit	T. Wolsey.

Centenary Prizes.

Class A	F. W. Robards.
Class B	J. T. Share-Jones.
Class C	C. E. Rix.
Class D	C. Radway.

CLASS PRIZES.

Class D.

MEDICINE.—*Silver Medal*—T. Wolsey. *Bronze Medal*—C. Radway. *First-Class Honour Certificates*—W. H. Anderson, C. W. Abrams, G. W. Bloxsome, E. Brown, R. W. Carless, A. H. Cory, H. Gamble, W. H. Hirst, E. O. Lander, J. W. Oakley, E. Peacey, C. F. Parsons, G. J. Roberts, P. J. Simpson, J. B. Walker, H. A. Woodruff, W. R. Williams, A. Whicher,

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Second-Class Honour Certificates—W. H. Brown, J. W. Conchie, W. Cranston, T. Elliott, L. W. Heelis, H. W. Percy, H. T. Ryan, P. W. Smith.

SURGERY.—*Silver Medal*—H. A. Woodruff. *Bronze Medal*—C. Radway and T. Wolsey (æq.). *First-Class Honour Certificate* — H. A. Woodruff. *Second-Class Honour Certificates*—C. W. Abrams, G. W. Bloxsome, E. Brown, J. W. Conchie, R. W. Carless, A. H. Cory, W. Cranston, H. Gamble, W. H. Hirst, E. O. Lander, J. W. Oakley, E. Peacey, H. W. Percy, G. J. Roberts, P. J. Simpson, J. B. Walker, W. R. Williams, A. Whicher.

Class C.

MATERIA MEDICA.— *Silver Medal*—G. Lockwood. *Bronze Medal*—J. J. B. Tapley. *First-Class Honour Certificates*—A. M. Brodie, H. Burrell, W. Lawson, J. H. Poles, D. J. Quinlan, C. E. Rix, C. Roberts, J. M. Tate, G. H. Wooldridge. *Second-Class Honour Certificates*—H. G. Allen, H. H. Jeffries, A. L. Purdy, E. L. Siddall.

HYGIENE.—*Silver Medal*—J. M. Tate. *Bronze Medal*—C. E. Rix. *First-Class Honour Certificates*—G. Lockwood, C. H. Rait, J. J. B. Tapley, G. H. Wooldridge. *Second-Class Honour Certificates*—H. G. Allen, J. Cane, J. C. Munby, J. H. Poles, D. J. Quinlan, C. Roberts, C. M. Sharpe, E. L. Siddall.

PATHOLOGY.—*Silver Medal*—J. J. B. Tapley. *Bronze Medal*—C. E. Rix. *First-Class Honour Certificates*—G. Lockwood, J. M. Tate, G. H. Wooldridge. *Second-Class Honour Certificates*—H. G. Allen, H. Burrell, J. Cane, H. H. Jeffries, W. Lawson, J. H. Poles, A. L. Purdy, D. J. Quinlan, C. Roberts.

Class B.

ANATOMY. — *Silver Medal* — J. T. Share-Jones. *Bronze Medal*—H. Thackeray. *Second-Class Honour Certificates*—R. J. Collings, E. J. Wadley.

HISTOLOGY.—*Silver Medal*—H. Thackeray. *Bronze Medal.*—J. T. Share-Jones. *First-Class Honour Certificates*—H. H. Aldred, F. E. Mason, J. Nicholas, E. J. Wadley. *Second-Class Honour Certificates*—D. A. Aitchison, G. H. Broad, H. Cooper, R. J. Collings, J. A. Dixon, E. S. Gillett, K. Hewlett, S. Jethiji, G. May, C. Tyler, C. W. Wilson.

PHYSIOLOGY.—*Silver Medal*—J. T. Share-Jones. *Bronze Medal*—R. J. Collings. *Second-Class Honour Certificates*—H. H. Aldred, G. H. Broad, H. Cooper, J. A. Dixon, E. S. Gillett, H. Greenfield, S. Jethiji, A. Leaning, F. E. Mason, J. Nicholas, H. Thackeray, C. Tyler, E. J. Wadley, C. W. Wilson.

Class A.

CHEMISTRY AND TOXICOLOGY.—*Silver Medal*—F. W. Robards. *Bronze Medal*—W. T. Olver and W. E. Schofield (æq.). *First-Class Honour Certificates*—J. Adamson, J. T. Angwin, E. P. Argyle, W. H. Chase, A. A. Donelly, J. Harrison, A. Littlejohn, S. F. G. Pallin, E. C. Webb. *Second-Class Honour Certificates*—A. W. Allen, J. R. Baxter, G. S. Bruce, G. Crowhurst, J. L. Cooper, D. L. Davis, A. Edgar, H. H. S. George, L. G. Gryspeerdt, A. W. Hodgson, F. Hopkin, H. D. Jones, P. J. L. Kelland, W. G. Litt, R. Morford, R. Paine, E. M. Perry, G. H. Rainforth, A. G. Saunders, A. H. Wilkins.

PRACTICAL CHEMISTRY.—*Silver Medal*—R. E. Klyne. *Bronze Medal*—W. E. Schofield.

BIOLOGY.—*Silver Medal*—F. W. Robards. *Bronze Medal*—E. C. Webb. *First-Class Honour Certificates*—E. P. Argyle, A. Littlejohn, W. T. Olver,

S. F. G. Pallin. *Second-Class Honour Certificates*—J. Adamson, A W. Allen, J. T. Angwin, B. H. Benson, G. S. Bruce, W. H. Chase, J. L. Cooper, A. A. Donelly, A. Edgar, H. H. S. George, L. G. Gryspeerdt, J. Harrison, A. W. Hodgson, F. Hopkin, H. D. Jones, P. J. L. Kelland, W. G. Litt, G. H. Rainforth, A. G. Saunders, W. E. Schofield.

MINOR ANATOMY. — *Silver Medal* — F. W. Robards. *Bronze Medal*— E. C. Webb. *First-Class Honour Certificate*—S. F. G. Pallin. *Second-Class Honour Certificates*—J. Adamson, J. T. Angwin, E. P. Argyle, G. S. Bruce, W. H. Chase, H. H. S. George, A. W. Hodgson, A. Littlejohn, W. T. Olver, E. M. Perry, W. E. Schofield.

PASS LIST.

The following are the Pass Lists [1] of this Institution for Session 1897-98.

FIRST PROFESSIONAL EXAMINATION.

Messrs W. G. Evans, J. G. Fitzgerald, R. E. Mosedale, R. J. Sargent, T. W. H. Thomas, S. H. Howard-Jones, G. May, C. E. Turtill. *H. E. Whitmore, S. J. Young, J. Adamson, J. G. Angwin, E. P. Argyle, J. R. Baxter, G. S. Bruce, G. Crowhurst, W. H. Chase, J. L. Cooper, D. R. Davis, H. H. S. George, L. G. Gryspeerdt, J. C. Hally, J. Harrison, A. W. B. Hodgson, F. Hopkin, H. D. Jones, P. J. B. Kelland, †A. Littlejohn, E. S. Oliver, *W. T. Olver, *R. Paine, *S. F. G. Pallin, E. M. Perry, †F. W. Robards, A. G. Saunders, W. E. Schofield, E. C. Webb.

SECOND PROFESSIONAL EXAMINATION.

Messrs R. Burt, C. R. Copp, F. W. Cousens, C. H. Coombe, *J. W. Flanagan, *C. G. Hearn, G. T. Jackson, B. L. Lake, *G. V. McNaboe, *J. C. Munby, H. Moseley, *C. E. Steel, †A. Spicer, *D. A. Aitchison, H. Anthony, *H. H. Aldred, G. H. Broad, *P. V. Beatty, H. Cooper, †J. A. Dixon, *E. S. Gillett, *K. Hewlett, A. Leaning, E. Nicholson, W. S. Stevens, E. Child, P. T. B. Bassett, *R. J. Collings, W. G. Flanagan, H. Greenfield, *The Kumar Shri Jethiji, *F. E. Mason, *H. A. Reed, *J. T. Share-Jones, *C. Tyler, †H. Thackeray, *G. Tucker, *L. M. Verney, C. W. Wilson, *E. J. Wadley.

THIRD PROFESSIONAL EXAMINATION.

Messrs *E. A. Batt, R. C. Bell, D. Crole, O. S. Fisher, A. Hudson, C. H. Joliffe, F. H. Ingersoll, *J. M'Leod, T. J. Symes, *A. M. Brodie, J. Cane, *H. S. Elphick, H. H. Jeffries, *G. Lockwood, W. Lawson, *J. Lee, W. R. Neale, F. S. Probyn, J. H. Poles, A. L. Purdy, *D. J. Quinlan, *C. E. Rix, D. H. Rait, C. Roberts, C. M. Sharpe, E. L. Siddall, J. J. B. Tapley, J. M. Tate, *G. H. Wooldridge, H. Fernandez.

FINAL EXAMINATION.

Messrs F. Crowhurst, Maidstone; R. C. Cochrane, Starbane, Ireland; F. C. Hobbs, Newnham-on-Severn, Gloucestershire; A. S. Head, London; G. H. Kitchin, Scarborough, Yorkshire; *P. Lloyd, Southport; H. O'Neill, Dublin; C. M. W. Park, London; W. G. Taylor, Hungerford; W. Murts, London;

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[1] In this and the succeeding Pass Lists † indicates with First-Class Honours, and * with Second-Class Honours.

F. W. Trydell, Groombridge, Sussex; J. H. Hulseberg, London; H. M. Durrant, Newcastle-on-Tyne; R. S. Collihole, Wareham, Dorset; C. W. B. Sikes, Lincoln; A. N. M. Swanston, Ealing; W. G. Green, Bournemouth; M. Glasse, Guernsey; W. H. Anderson, Ennis, Co. Clare; C. W. Abrams, Slough, Bucks; J. Buckingham, Harleston, Norfolk; W. H. Brown, Northampton; *G. W. Bloxsome, London; *E. Brown, Newtownards, Co. Down; J. W. Conchie, Wolverhampton; *R. W. Carless, Stafford; A. .H. Cory, Bideford, N. Devon; E. T. Ensor, London; T. Elliott, Durham; W. H. Hirst, Salford; L. W. Heelis, Handsworth Wood, near Birmingham; *E. O. Lander, · Shifnal, Shropshire; *J. W. Oakley, Uppingham; H. O. Oliver, Birmingham; E. Peacey, Tewkesbury; H. W. Percy, Athlone, Ireland; *C. F. Parsons, Athlone, Ireland; †C. Radway, St. Johns, London; G. J. Roberts, Gaerwen, Anglesey; H. T. Ryan, Dublin; P. W. Smith, Hendon, Middlesex; J. B. Walker, Maidenhead, Bucks; W. M. Williams, Llanwrda, S. Wales; H. A. Woodruff, Sheffield; *T. Wolsey, Lee, Kent; W. R. Williams, Nantymoel, near Bridgend, S. Wales; A. Whicher, Milford Haven, Pembrokeshire; E. A. L. Fenner, Shoreham, Sussex; F. W. Cox, Marshwood; G. F. Gould, Southampton; R. L. Green, Dudley, Worcestershire.

ROYAL (DICK) VETERINARY COLLEGE, EDINBURGH.

PASS LIST.

The following are the Pass Lists of this Institution for Session 1897-98.

FIRST PROFESSIONAL EXAMINATION.

Messrs K. Barker, G. E. Anderson, P. Braid, W. T. Dunstan, *W. Harris, W. A. Jellart, J. S. Nimmo, J. P. O'Reilly, J. Warwick, T. B. Redding, T. J. Keall, W. Nairn.

SECOND PROFESSIONAL EXAMINATION.

Messrs J. R. Atkins, N. Roberts, T. Fleming, W. J. Lewis, F. W. Paulett, *J. L. Still, G. Sykes, †W. J. Boyd, J. E. Cockroft, *C. Cunningham, *F. H. Cundell, R. A. Edwards, E. A. Ryan, J. Forrest, *G. Garden, F. Harper, *W. Hepburn, †R. B. Littler, T. Nunan, *S. E. Sampson.

THIRD PROFESSIONAL EXAMINATION (Four Years' Course).

Messrs E. Evans, W. J. Foley, J. L. Flood, J. W. Pollock, J. Maguire, P. Maher, J. O'Brien, H. E. A. Charles, F. W. Coats, W. P. Cushnahan, *R. Garraway, J. Garside, W. Girvan, A. Gofton, P. Hynes, J. F. Joyce, *W. S. King, *F. Leech, *E. Little, *G. H. Livesey, J. A. Macgregor, *J. McIlvenna, H. V. Mossman, J. O'Connor, E. O'Neill, *A. Robb, J. A. Russell, T. Hogg.

FINAL EXAMINATION.

Messrs. M. Howe, J. B. Kay, J. E. Holroyd, T. H. Sherlock, W. Scott, J. Watson, A. C. Burton, D. R. Chalmers, *A. Gibson, R. W. Hadfield, T. E. Loughran, *C. A. Read, W. J. Tart, C. E. Orton, F. Fail, A. Hodder, T. D. Lambert, R. H. Lambert, A. M'Gown, D. Dudgeon, A. T. Williams.

GLASGOW VETERINARY COLLEGE.

PASS LIST.

The following are the Pass Lists of this Institution for Session 1897-98.

FIRST PROFESSIONAL EXAMINATION.

Messrs J. M'Cutcheon, T. M'Allinuey, H. G. Rodgers, J. Dawskin, A. P. Logan, *A. M'Dowell, *W. H. Edwards, J. H. Lyons, Arch. Logan, J. H. M'Laren, W. M. Ferguson, C. C. Muat.

SECOND PROFESSIONAL EXAMINATION.

Messrs J. Baird, T. Kilpatrick, G. H. Barber, W. G. Forbes.

THIRD PROFESSIONAL EXAMINATION.

Messrs C. W. Mackay, W. W. Lang, J. Stevenson, W. A. Kilpatrick, P. M'Kinlay, W. Watson, R. Porteous, J. Aitken, J. Montgomery.

FINAL EXAMINATION.

Messrs J. Marshall, J. Donaldson, T. Rennie, R. G. Anderson, W. A. Campbell, J. Paterson, T. G. Palgrave, M. T. Gibbin, *J. J. Smith, J. C. Erskine, J. Crawford, A. Fraser, D. S. Weir, R. G. Jones, *W. Crawford, D. S. Jack, W. Neilson, J. G. Reynard, F. Waddington.

NEW VETERINARY COLLEGE, EDINBURGH.

PASS LIST.

The following are the Pass Lists of this Institution for Session 1897-98.

FIRST PROFESSIONAL EXAMINATION.

Messrs H. Barrow, R. E. Harman, J. Latta, W. D. Lindsay, C. Pitt, R. Scott, *M. Dowerbycoe, F. J. Dunning, W. A. Elder, R. Finch, H. Kauntze, *H. Kirby, Z. B. Rutherford, F. Sugden, J. D. C. Ward, L. W. Lloyd, G. T. Anderson, †G. T. Willows.

SECOND PROFESSIONAL EXAMINATION.

Messrs R. Connochie, A. Scotson, E. Finnucane, C. Harrison, *T. Parker, J. W. Bee, S. Drabble, *E. Ashworth, S. C. Binks, W. G. Burndred, *F. Crossley, J. P. Dunphy, *H. E. Martin, A. M'Farlane, *T. Runciman, *B. Runciman, A. Spreull.

THIRD PROFESSIONAL EXAMINATION.

Messrs J. C. Blake, J. Paul, G. A. Harrison, G. Barras, *J. Evans, W. L. Gascoyne, D. M'Donald, *A. M'Nae, R. Plunkett, *H. Taylor, C. R. Twist, H. Dyson.

FINAL EXAMINATION.

Messrs *H. H. Ferguson, A. M. Porteus, J. W. Brittlebank, W. Jowett, A. S. Lawrie, A. O. Leary, E. Marrison, H. M. Webb, R. J. Bailey, M. J. Mitchell, T. C. Howatson, B. D. Williams, F. C. Fountain, G. E. Hamilton, H. Whipp, G. H. White.

ROYAL VETERINARY COLLEGE, LONDON.

INAUGURATION OF THE WINTER SESSION, 1898-99.

THE Winter Session of the Royal Veterinary College was opened on Monday, 3rd October. The inaugural address was delivered by Professor Shave, and there was a large attendance of Governors, prominent members of the veterinary profession, and students.

At the outset of the proceedings Principal M'Fadyean said : " He was sorry to say that they had received a letter from Major-General Sir Henry Ewart expressing his regret that he was unable to take the Chair on that occasion as arranged. There was, however, one circumstance that tended to make their regret perhaps not quite so sharp as it would otherwise have been. They happened to be honoured with the presence of the President of the Board of Agriculture amongst them (applause), and he had great pleasure in moving that he be asked to take the Chair " (applause).

The RIGHT HONOURABLE WALTER LONG, M.P., President of the Board of Agriculture, thereupon took the Chair, and said :—" Gentlemen, I am sure that you will all agree with me in regretting very much the absence of Major-General Sir Henry Ewart, whose presence would have been very welcome here, and I am very conscious that I shall prove but a very indifferent substitute for him, but I shall endeavour to conduct the proceedings in an orderly manner, and according to the programme which has been placed before me. I do not intend to trouble you with any remarks of my own at this stage of the proceedings, but shall immediately call upon Professor Shave to read his introductory address " (applause).

PROFESSOR SHAVE. F.R.C.V.S., who was received with applause, then delivered the introductory lecture. He said :—

" Mr Chairman, my Lords, and Gentlemen. By the courtesy of our Principal I have been called to this position, and, to commence with an old saying the truth of which I have often felt, ' there is nothing so difficult as a beginning.' The great object of this day is to help our young friends, the new pupils, to make a good start, and it is most helpful and gratifying to us the teachers of this Institution to be surrounded and supported on these occasions by so many sympathetic friends.

" Many members of the profession embrace the opportunity afforded by this opening day of the session to manifest the interest they feel in the welfare of the old place, and of meeting each other and looking around for alterations and improvements which the Governors are making from time to time in this great nursery of veterinary science.

" Let me make a beginning by offering my most hearty congratulations to you, gentlemen, who have just entered this school and are about to commence your studies as veterinary students.

" It is generally said that a student's life in London is beset with sore temptation. You will find all the temptations easy to bear if you work hard, and this is necessary if you wish to leave here with your qualification at the end of the four years. My advice to you is be attentive to all the instruction you will get here during the day, and give three or four hours each evening to quiet reading and meditation in your rooms, go in for some reasonable form of recreation on Saturday afternoon and evening, and rest on the Sunday. You will find this enjoyable and necessary after the week's work. This prescription may sound rather hard, but one cannot expect a handsome reward without work, and you must look upon the four years you will spend here as your preparation for life, and be assured it depends very largely on how you spend your time here as to whether you will be a success or failure in the world.

" During the first year your studies will include chemistry, biology, anatomy, and manipulation of animals, and if your energy and perseverance are found equal to the examination you will pass on and apply the knowledge acquired to the study of anatomy, physiology, and histology, which will together with practical details of stable management constitute your second year's work. These subjects constitute the base of your pyramid—of medical and surgical knowledge. You have all heard of the advantage of a concrete foundation and the results which are likely to happen to houses built upon the sand. Take every opportunity of familiarising yourselves with the naked eye and microscopic appearances of the healthy organs and tissues of the body, for they are to form your standard of comparison.

" In the third year you begin your study of disease, and if you approach it well equipped with a sound foundation, that is, a knowledge of all the preceding subjects, you will find pathology not only intensely interesting but comparatively easy. You will also during this year be instructed in the actions and uses of medicines as applied to the cure or relief of disease, together with the proper construction of stables and other habitations of animals, as well as the best way of feeding animals under all sorts of conditions. In the fourth year you will learn to apply the information which you have acquired during the preceding sessions and be generally instructed in the practice of veterinary medicine and surgery, including the practical use of anæsthetics. This being accomplished there only remains the final examination before admission into the profession.

" And let me advise you, do not at first nor until you have your qualification cultivate a preference for any particular branch of your study ; the curriculum has been evolved for you and is the result of the thought and experience of generations. Do not think then for a moment that any part of it is superfluous or unnecessary, start with the idea of allowing yourselves to be taught, don't imagine that I mean you are to be passive receptacles, you are most certainly to think for yourselves. Look upon your teachers as your friends, bring your difficulties to them, and be assured it will give them great pleasure to assist in relieving your burdens. Bring your reason to bear on each subject and trust as little as possible to mere memory. You should carefully read the rules of the Institution, which you will find with a considerable amount of other useful information concerning the course in the Calendar, a copy of which will be presented to each of you.

" It is usual for each teacher at his first lecture to make a few general remarks on his course of instruction and give you the necessary information as to books and other details; don't therefore be absent from the first lecture. There is one book that you will require at the outset—a good medical dictionary, for although you have passed a very fair educational examination you will hear and read in the sciences many words altogether new to you. Never pass over such words or guess at their meaning, but look them up at once.

" Just one other word, cultivate the habit of punctuality. It will be of great use to you in business, and it is after all just as easy as being late ; being always late is only a habit and a bad habit, therefore make up your minds at once, always be in good time, it requires no greater haste, haste, which generally means flurry, is always bad form in a professional man.

" There are many prizes offered for competition among you by and through the medium of this College, some of very considerable intrinsic value. All of them should be eagerly sought after, every student should strive to gain something in competition with his fellows, for believe me there is much pleasure in success. Most of the prizes are confined to the students of this school, but there are some of much higher value because they are open to all students of veterinary medicine. I refer to the prizes offered by the munificence of Sir Frederick Fitzwygram, Bart., and it should be the ambition of you all, and I

say this to all veterinary students, to work for these prizes not only for your own honour but for the credit of the school to which you belong. I have just a few observations which I should like to make to those students who will shortly, perhaps before the next opening day, enter the profession.

"You will soon be admitted to the profession of your choice, and I think one may say that with a fair amount of industry, coupled with a good moral character, you are tolerably certain of making a moderate living. It is not a profession in which there are many large money prizes, but on the other hand there need be no blanks; it is a profession which teems with interest for all who love animals and desire to alleviate their sufferings. Be gentle and kind to your patients; never give them any unnecessary pain; do not tolerate cruelty in any form; it is frequently necessary to restrain our patients, but it can often be done without the infliction of pain; remember that the application of the oftimes necessary 'twitch' is very painful; use it only when other and milder means are inefficient and not as a routine measure. In all your operations consider the suffering of the animal, and whenever advisable either by the duration of the operation or the acuteness of the pain use an anæsthetic. With the practical instruction you have had here in the administration of anæsthetics, they can be used with sufficient safety to warrant you in employing them more frequently than has perhaps hitherto been the case; the little extra time and expense must not be considered when the suffering of the animal is at stake, and, further, the danger to the animal is minimised both as regards shock and muscular efforts, which latter occasionally lead to broken limbs or back, a matter of no small consideration to the young practitioner, and lastly there is the greater comfort to the operator, who knowing that his cuts are painless can be more deliberate and precise. You have been well drilled in bacteriology and in the use of antiseptics; bring your knowledge of these subjects to bear on your surgical practice; the aseptic treatment of wounds is one of the blessings of the century.

"Wounds which before its introduction were intensely painful are now practically painless. I know that aseptic surgery is difficult in our animals, but it is often possible and might be much more often practised. However, things in this respect are improving, for veterinary surgeons no longer look upon pus as necessary to the healing of wounds, though it is still much too often seen; look upon its presence in your operation wounds, gentlemen, as a reflection on your skill.

"On being admitted to membership you should each carefully read and keep the rules of the Royal College of Veterinary Surgeons, by which on signing the Register you agree to abide, and you should look upon yourself as a representative of a small but important profession which is trying, and one may say succeeding, in raising its head to the level of other professions, but we can only do this by the co-operation of each member. The progress of the profession and the confidence of the public must be our first considerations, and it is in the power of each member to do much to help this onward movement. Try to earn the respect of your fellow citizens, not only for yourself but for your profession; take our friend Mr Hunting's advice, get seats on the Local Governing Boards and teach the members the need of the services of the profession. See to your own interest, see that you get your rights, rather than grumble at your wrongs. Treat your professional brethren as you would have them treat you. Do not attempt to push yourselves by unseemly advertisement in the public press, or by even more objectionable, because less honest, methods to which it is to be regretted some members have stooped. Your council like the governing bodies of other professions have decided that advertisement of this kind is prejudicial to the best interests of the profession. In short, you will no doubt agree with me

that as we have made up our minds to be recognised as a profession we must not consider it a hardship to do as other professions do. One word in conclusion, don't forget you must always remain students; read the current professional literature and as many new books as time permits; take an interest in and attend some Veterinary Medical Association, or you must of necessity fall out of the ranks and perhaps become a fossil during life.

"Now, gentlemen, having finished my remarks to our young friends I thought, perhaps, a few observations on one or two subjects of interest, not only to the profession but to the public, would be suitable to the occasion.

"In our evolution we have received much from those whom we may consider as the leading scientific lights of the world; above all, because he was the pioneer, we think of Pasteur. His work is so well known and appreciated that one need only mention his name. Then Dr Koch, whose name is associated with a bacillus, the cause of tuberculosis, and with tuberculin, a product with which we hope to reduce or perhaps in time practically eliminate tuberculosis in cattle. It is to tuberculosis in some of its relations to Public Health and to its diminution in cattle that I would draw your attention for a few minutes.

"The first point to be established is the prevalence of the disease in cattle, and especially dairy cows in this country.

"That the disease is very prevalent, especially in dairy cows, is, I think, one may say the unanimous conclusion to which veterinary surgeons as a result of their experience have been driven. No doubt precise statistics would be more convincing, and the profession may in the near future provide these if the veterinary surgeons throughout the country will use tuberculin and record their results. Speaking to an audience like this, one might have taken the prevalence of the disease in cattle for granted, but one is warned against this course by the expression of opinion which has lately found vent in *The Times* newspaper, in which it was sought to remove one co·ner stone of the argument for the general use of tuberculin.

"There is, however, much precise evidence which can at the present time be brought forward in support of the frequency with which this disease affects cows. In this short sketch I can only mention two or three representative examples. Thus of 300 cows slaughtered in Edinburgh in 1890, 40 per cent. were affected (*vide* Report, Royal Commission on Tuberculosis, 1895); of 1109 cows tested with tuberculin in different parts of England, by twenty-seven veterinary surgeons, 426 gave the reaction, that is, 38·4 per cent. (*vide* Report of Principal M'Fadyean to Royal Agricultural Society, 1897), and, says the same authority, there are no grounds for believing that this represents an exceptional state of affairs for this class of animal, namely, dairy cows. Just one other instance from The Report of Royal Commission on Tuberculosis, 1898—of ninety cows tested with tuberculin in Cheshire, 77 per cent. reacted, and of six herds in the same neighbourhood 63 per cent. were found affected. In this place one should also note that the disease is more frequent in some districts than in others, and that some herds tested with tuberculin have been found quite free from the disease.

"As to the identity of the disease in men and animals, on this point there seems no difference of opinion. The Royal Commission on Tuberculosis which reported in 1895 entertained no doubt as to the identity of the disease in men and animals, and that it is capable of communication from men to animals, and *vice versa*. Further, that any person who takes tuberculous matter into the body as food, incurs risk of acquiring the disease. The Commission of 1898 have not had any doubts raised in their minds as to the accuracy of this opinion.

"Now, as to the human death-rate, at all ages, from tuberculosis, the Royal

Commission on Tuberculosis, 1898, notes with satisfaction that death from tubercular disease in all its forms and at all age periods has steadily fallen from 3483 per million during 1851-60 to 2122 during 1891-95, a diminution equal to 39·1 per cent. This diminution applies to all age periods, but not equally.

"For the age period 0-5, the only one to which I would particularly call your attention, there has been a reduction in the death-rate during the last thirty years from tubercular disease in all its forms equal to 27·9 per cent.

"But if we examine Tabes mesenterica at this age period, we find the diminution is only equal to 3 per cent.

"At this age milk is the principal diet of the child, and Tabes mesenterica is generally considered to be produced by the ingestion of tubercular matter. During the same period (last thirty years) there has been a considerable increase in the consumption of milk per head of the population. The point to be noticed is that all other forms of tubercular disease during the milk-drinking age have notably diminished except the one which, by general consent, is considered most likely to be produced by ingestion. This directs our attention to the principal article ingested, namely, milk.

"Dr Woodhead found that out of 127 cases of tuberculosis in children, the mesenteric glands showed tubercular affection in 100, and that there was ulceration of the intestine in 43.

"Concerning milk : is there any direct evidence that milk contaminated with the tubercle bacillus is ever exposed for sale?

"Dr Hope, Medical Officer of Health of the City of Liverpool (*see* Report of Royal Commission on Tuberculosis, 46, 1898), has recorded the result of bacteriological examination of 144 samples of milk taken from sources within the city. The tubercle bacillus was detected in 2·8 per cent., and in 24 samples taken at railway station of milk arriving from the country the tubercle bacillus was detected in 29·1 per cent. Principal M'Fadyean told the Royal Commission, 1898, that in a sample of milk from a diseased udder, submitted to him for diagnosis, he had no difficulty in detecting the tubercle bacillus, yet the milk from that cow continued to be sent in for sale to a neighbouring city. Is uncooked milk containing the tubercle bacillus infective to animals fed on it ? Dr Martin, in his report to the Royal Commission on Tuberculosis, 1895, says in every instance the animals fed on milk taken from cows with tubercular disease of the udder, and in which the tubercle bacillus could be found on microscopic examination, acquired the disease. Now, as to the human subject, notice a case recorded by Ollivier at a meeting of the Paris Academy of Medicine (quoted by Almond in paper read before the National Veterinary Association, 1896), that in a young ladies' boarding school twelve girls presented symptoms of tuberculosis, and of these, five died. Since all the girls had healthy parents, and especially since all of them had well-marked intestinal tuberculosis, the food was suspected. It was then found that the cow that had for years supplied the school with milk was tuberculous, and the autopsy showed that not only the cow's viscera, but the udder was badly affected. Both the Royal Commissions are unanimous on this point, viz., that milk taken from cows with tubercular disease of the udder is infective to animals and mankind.

"Are tubercular animals with healthy udders a safe source of milk supply ? Dr Martin, in his report to the Royal Commission, 1895, says forty-one animals fed on the milk of eight tuberculous cows which had healthy udders remained free from tuberculosis, and twenty-eight animals inoculated with their milk also remained quite free.

"In a tuberculous cow, can we be certain that the udder is healthy? What was the opinion of the Royal Commission on Tuberculosis, 1895, referring to udder disease? They say 'the very absence of any definite sign in the earlier stage is one of the greatest dangers of the condition.' We must

acknowledge that there is a stage in tubercular disease of the udder at which we could not diagnose it, but even if we could say that this udder is healthy, are there not still the facts that the disease may extend to the udder at any time, that when it does so it rapidly extends, and, of course, infects the milk?

"Must we not, then, agree with Drs Martin and Woodhead, who 'insist that no tuberculous animal should be allowed to remain in a dairy?' Probably many of us would; but we must also agree with the Royal Commission which has just reported, that, having regard to the number of dairy cows affected, the elimination of tuberculous cows from dairies must be done gradually, and if only the suggestions made with regard to inspection of dairy cows by veterinary surgeons, and powers for the Medical Officers of Health to stop the milk supplied by diseased animals on the certificate of a veterinary surgeon, are carried out, the public may congratulate themselves on their comparative safety.

"One other point worth noticing is that Drs Woodhead and Martin assert that butter, butter-milk, and skimmed milk obtained from the milk of a cow having a tuberculous udder all contain infective matter, and are actively injurious to test animals.

"Pertaining to legislation in England as to the sale of milk contaminated with tubercular matter the points to be noticed are: Tuberculosis is not included in the Contagious Diseases (Animals) Act, 1878, and further, referring to milk drawn from a cow with tubercular disease of the udder, the Royal Commission on Tuberculosis (Note 38, 1898) say there is no power at present to prevent such milk being sold.

"One may sum up this part of the subject in a few words. That tubercular disease is prevalent in dairy cows in this country.

"That milk drawn from cows with tubercular disease of the udder does contain tubercular matter (bacilli), and if taken uncooked as food is infectious, and does produce the disease in animals and mankind.

"That milk containing tubercular matter is exposed for sale and sold for human consumption.

"That the manifestation of tubercular disease in children (*Tabes mesenterica*) is generally believed to be caused by the ingestion of tubercular matter.

"That this manifestation of tubercular disease (*Tabes mesenterica*) has not diminished in anything like the proportion of its other manifestations during the milk-drinking age, thereby pointing to milk as a cause.

"That there is at present no effective power to prevent the sale of milk though it may be known to contain tubercular matter.

"Now, anomalous as it may seem, there is legislative power to prevent the addition of water, even pure water, to milk, but none to prevent contamination with a poison from which it is said one-seventh of mankind die, and from which innumerable children suffer for years, and if they at length recover it is often after numerous surgical operations, many of them of necessity disfiguring and often leaving the little patients cripples for life.

"One can only conclude that the public do not yet appreciate the risk they incur by feeding their children on uncooked cow's milk, or possibly we should hear more about milk contamination and maybe less about the fancied evils of dog-muzzling and vaccination.

"Having satisfied ourselves that tuberculosis is prevalent in cows, and a source of danger to the public, and pointed to the remedy as contained in the Report of the Royal Commission on Tuberculosis, 1898, it goes without saying that this disease occasions great loss to the agriculturalists of this country.

"Can we, as veterinary surgeons, do anything to lessen this evil? We can now answer this question with confidence in the affirmative, since the introduction of Koch's tuberculin, and its use for diagnostic purposes by

Dr ·Bang of Copenhagen and many continental veterinary surgeons, by Principal M'Fadyean and numerous veterinary surgeons in this country, and further, since opinion as to its diagnostic value is unanimous. Just a few words in support of this assertion. The Royal Commission on Tuberculosis, 1898, says : 'We entertain no doubt as to the value of tuberculin, provided the test is applied by a competent veterinary surgeon, and that the tuberculin is of trustworthy quality.' Dr Bang, whose experience is enormous, says tuberculin is reliable in over 90 per cent.

"Principal M'Fadyean has most implicit faith in tuberculin as a test for tuberculosis in animals when standing in their own premises and undisturbed. It is not reliable when used on cattle in a market-place or otherwise excited.

"We have, then, at our command a reliable diagnostic agent which will enable us, if we use it according to a few simple rules now well understood by the profession, to separate diseased from healthy animals. This is a great step ; and it seems to be the duty of the veterinary surgeon, having assured himself of the value of this agent, to do all in his power to recommend its use to the cow-keeping public.

"As to checking the spread of, and possibly in time eliminating, tuberculosis from our herds, we have the valuable experience of Dr Bang of Copenhagen, whose methods have been attended with such splendid results in Denmark. You have no doubt read the interesting communication by Dr Bang to the International Veterinary Congress, which was translated for the *Journal of Comparative Pathology and Therapeutics,* 1895, also a paper dealing with this subject, read by Mr Almond before the National Veterinary Association, 1896. It will, therefore, only be necessary for me to allude in a general way to this matter.

"The initial step in dealing with this disease seems to be to recognise its contagious nature, to be well acquainted with all the details of the life-history of Koch's bacillus, and to be well informed as to the methods by which it is spread. Secondly, to make an early diagnosis, and, thirdly, to separate diseased from healthy animals—in other words, to deal with tuberculosis as you would with any other infectious disease.

"As to the first step, we have all recognised the contagious nature of the disease, and the facts contained in the following statement, taken from Muir and Ritchie's *Manual of Bacteriology,* that 'though the experiments of Sander show that the tubercle bacillus can multiply on vegetable media to a certain extent at warm summer temperatures, it is doubtful whether all the conditions necessary for growth are provided to any extent in nature. At any rate the great multiplying ground of tubercle bacilli is the animal body, and tubercular tissues and secretions containing the bacilli are the chief if not the only means by which the disease is spread.'

"We see then that tubercular tissues and secretions containing bacilli are really what we have to deal with, and to have these in close, dirty, badly-ventilated cow-sheds almost certainly means infection and spread of the disease. Whether the bacilli are inhaled or ingested matters little, hence the importance of the second step—early diagnosis, by which we can get rid of the diseased animals or sources of infection. This early diagnosis was, until the introduction of tuberculin, impossible ; now it is easy, harmless, certain.

"It is perhaps necessary to mention in this place hereditary predisposition, and to say there are, so far as cattle are concerned, no facts which warrant us in believing that this is an important factor in the spread of the disease. We should notice, however, that in some herds nearly all the members are diseased, which shows that under circumstances which favour infection nearly all cattle are, so to speak, predisposed to the disease, but whether this predisposition has anything to do with heredity may at present well be doubted.

"With reference to congenital tuberculosis, it undoubtedly occasionally

occurs in cattle. As to the frequency of its occurrence we have no statistics for this country. Dr Bang considers that about ·3 to ·4 per cent. of calves are affected at birth.

"With the exception of a few calves born with the disease all experience points to infection after birth, for the disease, comparatively rare in calves, progressively increases with age. Thus, to quote from Dr Bang's statistics, but omitting fractions, at six months old and less 15 per cent. are diseased ; at one year 30 per cent. ; at two years 40 per cent. We can only hope that the disease is not so prevalent in this country, but we have no sufficient statistical evidence on which to form any conclusion.

"There are people who assert that the disease is not so prevalent in this country as we believe it to be. Consequently statistics from those gentlemen having the opportunity of using tuberculin would be very valuable.

"Having by means of the tuberculin test separated the diseased from the healthy animals, the question arises, What shall become of the re-acting (diseased) animals? It would seem advisable to consider them in at least three groups :—

"1. Those with the udder affected are generally far advanced in the disease, are useless for dairy purposes, and should be disposed of; whether or not they are fit for meat the inspector will decide.

"2. Those with evident clinical signs of the disease, especially if associated with cough or discharges, which, of course, contaminate the sheds ; if not disposed of they should certainly be kept by themselves.

"3. Those which show no obvious signs of the disease. Many of these are only slightly affected, and under favourable circumstances some will no doubt recover, and when retested several months afterwards may not re-act to tuberculin. Whether or not these may be safely returned to the healthy herd is a question which experience only can decide.

"On the evidence one seems justified in sending their milk to market or using it in the dairy ; but one notices that the milk of re-acting animals is not used on the Danish farms for feeding calves, but is sent to the dairy (*vide* Report Royal Commission on Tuberculosis, 1898).

"As to breeding from re-acting animals, it would seem practically safe from Group 3. It could hardly be recommended in Groups 1 or 2. So long as the udder is healthy, there seems no reason why the calves should not be fed on the milk of Group 3. Greater safety would of course be ensured by boiling it. Calves born of diseased mothers should be separated as soon after birth as convenient, and not allowed to re-enter infected sheds.

"All young animals should be tested as soon as possible, and all fresh purchases should be tested before being admitted to the healthy herd. The healthy portion of the herd should be tested from time to time, as may be considered necessary.

"These are broadly the lines on which great success has been achieved by Dr Bang and others, with the assistance of the Danish Government, in reducing the disease in Denmark. And what has been done in Denmark, if we can secure assistance as recommended by the Royal Commission on Tuberculosis, 1898, one may venture to hope can be done in England.

"The question suggests itself. Should the agricultural community avail themselves at once, without waiting for government help, of this valuable, cheap, and harmless method of detecting the disease, and by the adoption of the simple methods check the affection? They must of course answer the question for themselves, and in this connection they might do well to consider that legislative measures, at least so far as the inspection of milk and meat are concerned, will probably not be long delayed, that the tuberculin test is likely to be soon generally recognised, and that buying and selling animals subject to it is likely to be the rule. That Government after all may not assist them, and

the Royal Commission on Tuberculosis were not unanimous in the matter of compensation.

"There are many other subjects on which one might have touched with advantage, indeed, on considering this matter I thought to have covered much more ground in the time. I wished especially to say something on the usefulness of the mallein test for glanders in horses and on the good work our young members are doing in South Africa in dealing with cattle plague, but I feel that already too much time has been occupied, and there yet remain several points which just require notice.

"As interesting to the general practitioner, one may observe the report by M. Flocard of Geneva, read by M. Chauveau to the Societé Nat. d'Agriculture de France, concerning the results of oöphorectomy or castration in cows, in which it is stated that cows so operated on give more milk, to the extent of 1300 or 1400 litres per annum, fatten more quickly, and that the meat is of better quality. The operation is done through the vagina with the animal standing, and seems in M. Flocard's hands to be without much risk to life, since between 1888 and 1897 he performed 2505 operations without an accident. If anything like this result could be obtained in England one would think the operation might become popular.

"Attention has been called by Dr Ewart, F.R.S. in an important monograph, 'A Critical Period in the Development of the Horse,' to a question of much importance namely, 'breaking service' or abortion in mares; and the question of artificial insemination of so-called barren mares has been introduced by Mr Walter Heape, M.A. in articles communicated to the *Veterinarian*.

"It is also gratifying to note on the authority of MM. Nocard and Roux that the organism of pleuro-pneumonia in cattle has at last been seen, and by a somewhat novel method artificially cultivated.

"Some valuable and welcome additions have been made to our literature during the year. We have now in Capt. Hayes' translation of the first part of Friedberger and Fröhner's *Veterinary Pathology* at least a portion of a work, the original of which (as the reviewer remarks in the *Veterinarian*) stands to-day without a rival in the German-speaking countries; also *A Handbook of Horse-shoeing*, by J. A. W. Dollar and Albert Wheatley. This important subject has at all periods of our evolution received the earnest attention of our most distinguished members, and books dealing with it are always in request. The subject had, in this College, as early as 1799, acquired many of the principles on which we now insist. Let me read you a short paragraph from the *Sun* newspaper, dated 26th October 1799, which was brought to my notice by Professor Penberthy.

"'Shoeing of Horses *as prescribed by the Veterinary College.*—Nothing to be cut from the soles, binders, or frog, but loose rotten scales. No shoes to be fitted on red hot. Shoes to be made of good iron, with a flat surface for the horn to stand on. Web not so wide as formerly, and weakest at the heel that the frog may rest on the ground. No more opening of heels.'

"There are several directions in which the profession may be complimented.

"We must all, I think, highly appreciate the recognition we have received at the hands of the Royal Commission on Tuberculosis, 1898, and we must, as a profession, do all in our power to justify the confidence of the Commission, which has proposed to call some of our members to most important public positions.

"In memory of the sixtieth anniversary of the reign of our beloved Queen, some members of the profession conceived the idea of a Victoria Veterinary Benevolent Fund, the rules and objects of which are known to all. It has received at the hands of many members most handsome support, and needs no words of mine to recommend it to all veterinary surgeons, to whom the success of this fund is a matter for sincere congratulation.

" There is just one other matter (last, but by no means least) on which certain gentlemen in particular, and in a general way the profession as a whole, are to be sincerely congratulated, namely, the honours which Her Majesty's Government has lately been graciously pleased to shower on us. The honour of knighthood has been conferred on Professor (now Sir George) Brown ; and we must all be proud to know that this distinction was bestowed on him in consequence of the professional services which he has rendered to his country through the Board of Agriculture. The other happy recipients of Royal favour are Professor Penberthy, F.R.C.V.S., and R. C. Trigger, Esq., J.P., F.R.C.V.S., each of whom has received, during his year of presidency, the medal to be worn in commemoration of the sixtieth anniversary of Her Majesty's reign. I am sure you will all recognise the merit of these gentlemen, and join me in offering our most hearty congratulations, coupled with our wishes that they may live long and happily to enjoy their well-earned honour. In conclusion, gentlemen, allow me to thank you for your presence, and for the patient way you have listened to my somewhat lengthy remarks."

At the conclusion of the address, which was listened to with close attention and provoked frequent applause,

PROFESSOR SIR GEORGE BROWN, C.B., said :—" Mr Chairman, my Lords and Gentlemen, very fortunately the task which has fallen to my lot is a very gratifying one, and it can be performed with very considerable celerity. Professor Shave has given us a very eloquent address. He has traversed a great deal of ground, but he has made one omission with becoming modesty, and that omission gives me the opportunity of calling your attention to the paramount importance of the subject which it is his duty to teach. Without some knowledge—indeed, without an intimate knowledge of the structure of a piece of machinery you can hardly begin to repair it, and you may depend upon it that you may devote all your spare hours to working in the dissecting room with an advantage to yourselves, which you will only realise in the course of your future career. You will be able to look at an animal as it stands before you and see plainly in your mind's eye the position of every part of its structure, and this will give you an amount of coolness and of confidence in all your operations and in all your clinical procedure that you cannot acquire in any other way. The study of anatomy, gentlemen, is a feature which commends itself to me particularly, for it is a subject on which you need have none of those eccentricities which are included under the head of opinions (laughter), very common things indeed, and so worthless as a rule that one constantly wonders why people take the trouble to utter them (renewed laughter). It has been remarked, I think by an old writer of the platonic school, that the gods who are above man have something whereof men do partake, as knowledge and intelligence. The beasts which are below man have also something of which men do partake, viz., growth and sense, but man has something of which neither the gods nor the beasts do partake— something which has given him all the trouble and caused all the confusion in the world, and those are opinions (laughter). But in anatomy, gentlemen, you need not say ' it is my opinion that this is a bone, or a muscle,' because we know it is, and I commend the study of anatomy as an exercise in that particular direction that you may be enabled to cast all your opinions to the wind and set yourself down upon facts. I feel quite sure, gentlemen, that you will accord Professor Shave a very hearty vote of thanks for the lecture which he has given us " (applause).

The vote was carried by acclamation.

PROFESSER SHAVE, said :—" Mr Chairman, my Lords and Gentlemen, I feel that I have already taken up too much of the time of this meeting, so I will just thank you, sir, and the meeting for the very kind way you have received me."

THE PRINCIPAL then read the list of prizes, medals, etc., awarded on the results of the examinations held during the previous session. He said that the mere reading of a prize list of course did not afford any indication as to the general success of the session. He would like to take the opportunity of saying that in one very important respect, the past session was probably the most successful in the history of the Institution, viz., in respect to the number of students who were successful in gaining their diplomas or in passing their examinations. In the July examination, which was the principal one of the year, 150 students presented themselves for the examinations of the Royal College of Veterinary Surgeons, and of that number 110 or 73 per cent. passed. When one took into consideration that the examination of the Royal College of Veterinary Surgeons as now conducted, really constituted a very searching test, he thought the result was highly creditable, and he thought that the teachers of the College may without immodesty claim some share of the credit of that result (cheers). He was quite sure, however, that all his colleagues would concur with him when he said that this gratifying result was mainly due to the unusual intelligence and diligence which the students themselves brought to bear upon their studies during the past year."

PROFESSOR CROOKSHANK, said :—" My Lords and Gentlemen, I have a very easy task to perform this afternoon. It is sometimes customary in proposing a royal toast to mention simply the name of the Queen—that is quite sufficient, and to say no more—and on this occasion, in rising to ask you to carry a vote of thanks to our Chairman to-day, it would only be necessary for me to mention the name of Mr Long and to sit down (cheers). But I am told that I must be conventional, and I must give reasons for the gratitude which we feel to our Chairman to-day. If I were to give you all the reasons I should detain you too long, I will therefore restrict myself to one or two. Whether we are agriculturists, or landowners, or veterinary surgeons, we all know how intimately agriculture is associated with the veterinary profession (hear hear). Gentlemen, I wanted to say that no Minister of Agriculture has ever done more, or wished to do more, than Mr Long has done for the prosperity of agriculture in this country, and we know that the prosperity of the farmer must necessarily be reflected upon the prosperity of the veterinary surgeon. Gentlemen, I often think that in these days, when we hear so much of inoculation and tuberculosis and all those subjects against which it is not for me to say anything, we are liable to overlook one of the greatest of the advances of the present century which was initiated entirely by your profession. We are apt to overlook the great work that was carried out against very formidable opposition in adopting the principle which is commonly known under the heading, 'the Method of Stamping out Animal Plagues.' That work, which started during the cattle plague in 1865, was carried out by the veterinary profession, and in that respect the veterinary profession was a long way ahead of the medical profession, because those principles which were embodied in the methods of stamping out animal plagues—methods of disinfection and of isolation are now in a modified form carried out very largely in dealing with the plagues which affect mankind. I don't think that the work of the veterinary profession has ever been sufficiently appreciated by the general public—the work, I mean to say, in initiating the system of stamping out animal plagues, and, among many other reasons, we welcome Mr Long here to-day, for the very loyal way in which he has carried out those principles which are recognised by veterinary surgeons as the best means for stamping out animal plagues (cheers). I might have imagined that I saw a slight look of depression on the face of some pathologists when Mr Long entered the room, and I can only explain that on the ground that he has been so successful in exterminating and keeping out of the country many of the animal plagues ; that the pathologists have been deprived

of a good deal of material for their researches. Well, gentlemen, there is one more reason to which I will allude, which makes us all give a very hearty welcome to Mr Long to-day, I am sure whether we are governors or whether we are members of the staff, or whether we are students, we must all appreciate this fact, that here we have an introductory lecture at our veterinary school and a Cabinet Minister thinks it worth while to be present and at a moment's notice to take the Chair. I think we may take this as evidence of the recognition of the great work that is being carried out by this school (cheers). I am bound to say as a governor, and I think it is the feeling of all the governors, that the work which is carried out by this college and within these walls is not sufficiently appreciated by the general public, but I think we may take it as an evidence that the work is beginning to be appreciated by the public when a leading public man is willing to come down and join with us in the opening of our session (applause). It is a just recognition, sir, for which we are deeply grateful (hear hear). Gentlemen, it only remains for me to propose— and I am sure it will be most heartily carried—a vote of thanks to the President of the Board of Agriculture for so kindly taking the Chair to-day " (cheers).

The resolution was carried with acclamation.

The Right Hon. W. H. LONG, M.P., said :—" Gentlemen, I am very much obliged to the proposer of this resolution for the kind terms in which he has introduced it to you, and equally to you for your warm reception of his words. I am quite ready to accept what he laid down, namely, that my presence here to-day is to be regarded as a proof of the interest taken by the outside world in your great profession and in this great college, because I am confident that it is the simple fact, that the interest taken in the work of your profession and in the life which members of your profession are preparing for, has been largely growing in the importance which people outside are now prepared to give to it. I listened with the greatest possible gratification and interest to the introductory address of Professor Shave, and I am very glad, indeed, if I may be allowed to say so, that he selected for the main portion of his address this very interesting and important question of the elimination and possible stamping out of tuberculosis. I believe that the veterinary profession can do a very great deal to forward this movement, apart from any action which it may be necessary for the Government to take. All that the Royal Commission recommended that Government should do was in the first instance to provide the tuberculin, comparatively speaking at a small cost, and in the second place to pay what may be necessary in order to secure that properly qualified veterinary surgeons should carry out the operation. Well, gentlemen, the Royal Commission estimated that the cost of the tuberculin would be about 3d. per dose, and the cost of the individual application of the test to the cow would be about another 2s. 3d., coming altogether to about 2s. 6d. per case. I think those were about the figures. Well, I do not want in any way to seek to evade the responsibility which must always rest on the executive Government in matters of this kind. I have said elsewhere what I am prepared to repeat here. If I can only be satisfied that there is a general desire to make use of these opportunities there will certainly be no unwillingness on my part to approach the Treasury and obtain the necessary consent for the expenditure. But I would like to impress this upon you. Some of you are now living actively in the world, and others of you are going to. If a remedy of this kind is as efficacious as science now tells us it is, surely it can hardly be necessary that the Government should step in for the payment, amounting to 2s. 3d. per cow, in order to induce the owners of horned stock in this country to take advantage of this discovery. The real truth of the matter is, I am confident, that those men who have got the

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knowledge which is of such immense value, the great scientific knowledge which enables them to say not only that it is a remedy, but also to give the reason for the faith that is in them, do not make sufficient use of the opportunities which are presented to them to infuse their neighbours and the public generally with their own confidence and their own belief. I am sure if you accept the advice given you by Professor Shave, and do your best to make these benefits known, then the public will take greater interest in discoveries like this one. I think, perhaps, the discovery with regard to tuberculin may not unfairly be described as one of the most important that has ever been made, because, if it is carried out, and if the success that is foretold for it follows upon these operations, then not only shall we eradicate the disease which has caused great loss to owners of stock in the country, but scientists tell us—and the news is welcome indeed—that simultaneously with the eradication of the disease amongst cattle would come the eradication of that terrible disease of consumption amongst human beings, which has brought sorrow to so many homes, and has so long been regarded as an incurable and unpreventable disease (applause). Therefore, we have a double object to work for. Gentlemen, I am not going to trespass longer upon your time. It gives me great pleasure to be here at your proceedings to-day, and to wish you all success and prosperity in the new session entered upon. I regret that circumstances have made it necessary that I should fill the Chair. We should all have been glad if Major-General Ewart had been present, but I am very glad to come here, and I hope that as long as I occupy the position I do I shall be able to attend here, and subsequently, in a private capacity, in order to give proof of the interest which I take in the great profession to which many of you belong, and to which many others hope to belong. If I can add one word to those weighty words already spoken by Professor Shave, I say this : I believe to the members of the veterinary profession there is nothing of more importance when they are starting on their new career than to make it their aim to do everything in their power to raise and maintain the standard of their profession throughout the country (applause). Great strides have already been made ; there yet remains room for improvement in some districts. Gentlemen, I know you have heard of the battle that a professional man has to wage when he gets the dual position of being adviser to his clients on the one hand, and on the other hand gets some public position which often places upon him the unpleasant alternative of either discharging his public duty and offending his client and therefore losing his custom, or failing to discharge his public duty. I would say respectfully to the members of this profession, present and future, whom I have the honour to address, that they will do well in their own interests, and in the truest interests of the great profession to which they belong or hope to belong, if they will do their utmost to win in that struggle and to put public interest always before private ones, however urgent or pressing they may seem to be, because if they can succeed in doing that they will be doing more by so doing to raise and level up the standard of their own profession than I believe they can do by any other means (applause). I am very much obliged to you, gentlemen, for the way in which you have been good enough to receive me here this afternoon, and most heartily do I wish prosperity to those young men who come here to educate themselves for what is one of the greatest and noblest professions in which it is possible for a man to be " (applause).

RELATION BETWEEN THE WEIGHT OF A HORSE AND ITS WEIGHT-CARRYING POWER.

By Veterinary-Major F. SMITH, F.R.C.V.S., F.I.C., Army
Veterinary Department.

THE work which horses perform is either to drag a weight or to carry
one, and an attempt to estimate the muscular effort these entail has
occupied my attention for some years.   So far as the question of
draught is concerned I limited my enquiry to a consideration of the
maximum muscular effort of the horse[1]; in the present communi-
cation the object has been to ascertain the effective and not the total
weight a horse should carry.

   Direct investigation having been found impracticable, the method
of enquiry adopted was as follows: The weight the horse was
capable of carrying was estimated by an independent observer whose
judgment could be relied on.   The animal was then weighed.   In
this way we ascertained the proportion which the estimated weight-
carrying power bore to the body weight, and, further, the judgment
of the observer was by this means submitted to a rigid test.

   As to the amount of reliance and responsibility which is extended
to experts in judging the weight a horse should carry, it should be
pointed out that in practice no attempt is made to estimate that
weight to less than 7 lbs.; this is the unit of weight adopted
by universal consent, and it requires some years of experience before
such knowledge can be obtained and skilfully applied.

   The total number of observations was 136, divided into two series,
A and B.

   Series A comprises light horses such as are used by a regiment of

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[1] Journal of Physiology, Vol. xix., No. 3, 1896.

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light cavalry, while those in the B series were heavy cavalry horses. The same expert was not employed in each series, and in this way the accuracy of the method was more severely tested.

In the first instance we must see how far the method of work was reliable, and this is best arrived at by testing the judgment of the experts who decided the weight the horses were capable of carrying. If their judgment is sound, we should expect to find that all horses of the same body weight had much the same weight allotted them to carry, and, further, that as the body weight increases the carrying capabilities should also increase.

If we now examine series A in this manner, and select only the largest groups, we find that it stands the test fairly well. Before giving the figures, it is necessary to point out that the balance on which these horses were weighed was not sensitive within 28 lbs.

There were thirteen horses each weighing 952 lbs. These were estimated to carry 161 lbs. (1 horse), 168 lbs. (8 horses), 175 lbs. (2 horses), 182 lbs. (2 horses). Mean 170 lbs.

There were eight horses each weighing 980 lbs. These were estimated to be capable of carrying 168 lbs. (3 horses), 175 lbs. (2 horses), 182 lbs. (3 horses). Mean 175 lbs.

There were ten horses each weighing 1008 lbs.; the estimate was as follows:—168 lbs. (2 horses), 175 lbs. (3 horses), 182 lbs. (5 horses). Mean 177 lbs.

There were ten horses each weighing 1036 lbs.; their estimate was:—168 lbs. (2 horses), 175 lbs. (1 horse), 182 lbs. (7 horses). Mean 178·5 lbs.

There were fourteen horses each weighing 1092 lbs.; there was considerable variation in the weight allotted them, but the mean came out at 192·4 lbs.

There were seven horses each weighing 1120 lbs.; and these were estimated to carry 182 lbs. (3 horses), 196 lbs. (3 horses), 210 lbs. (1 horse), or a mean of 192 lbs.

In series B the body weight was ascertained more accurately (within 7 lbs.), the result being that the total number in the group of any one weight became much smaller, while the number of groups was more numerous.

To reduce the number of groups I proceeded as follows: Under the group of 10 cwts. is included all body weights which fall short of 10 cwts., 1 qr., and under the latter are included all that fall short of 10 cwts., 2 qrs. and so on. The mean weight of the groups was then taken. Selecting only the larger groups, we find as follows:—

Five horses gave a mean body weight of 1015 lbs.; estimated carrying capability 182 lbs. (4 horses), 189 lbs. (1 horse). Mean 183·4 lbs.

Seven horses had a mean weight of 1049 lbs.; their carrying weight was as follows: 182 lbs. (2 horses), 189 lbs. (5 horses). Mean 187 lbs.

Eight horses having a mean weight of 1081 lbs. were estimated as follows:—189 lbs. (3 horses), 196 lbs. (4 horses) 203 lbs. (1 horse). Mean 194 lbs.

Six horses with a mean weight of 1099 lbs. gave as follows: 189 lbs. (3 horses), 196 lbs. (1 horse), 203 lbs. (2 horses). Mean 195 lbs.

Ten horses with a mean weight of 1133 lbs. gave as follows: 182 lbs. (1 horse), 189 lbs. (2 horses), 196 lbs. (6 horses), 210 lbs. (1 horse). Mean 194 lbs.

Six horses having a mean weight of 1156 lbs. gave, 189 lbs. (1 horse), 196 lbs. (3 horses), 203 lbs. (2 horses). Mean 197·17 lbs.

Eight horses having a mean weight of 1186 lbs. gave, 196 lbs. (4 horses), 203 lbs. (1 horse), 210 lbs. (3 horses). Mean 202·12 lbs.

An examination of the figures contained in these two series, shows that the judgment of the gentlemen employed to estimate the carrying power may be relied upon ; of course, absolute uniformity in the groups could not be expected. Neither of the experts had the least idea of the body weight of the horse, they were unknown to each other, and three years elapsed between series A and B. Their judgment in the matter was influenced solely by the make and shape of the animal. It is satisfactory, therefore, to find that my forecast is for all purposes correct ; viz., that body weight and carrying power are intimately related.

We are now in a position to consider the subject of this communication, viz., what weight should a riding horse be asked to carry ? It is obvious that one important factor governs the reply to this question, viz., the pace at which the horse has to carry its load. All the observations in series A and B presuppose the animal to be working at the fastest pace (excepting racing) required from horses, viz., hunting.

The extra weight to be carried by horses performing ordinary saddle work, either in a military or civil capacity, will be mentioned subsequently.

SERIES A.

No. of Observations.	Body Weight.	Estimated Weight-carrying Power.	Calculated Weight-carrying Power.
	lbs.	lbs.	lbs.
1	840	161	147·58
1	924	182	162·22
13	952	170·7	167·36
8	980	175	172·19
10	1008	177	177·11
10	1036	178·8	182·53
4	1064	192·5	186·92
14	1092	192·4	192·78
1	1108	182	194·68
7	1120	192	196·77
1	1136	196	199·60
2	1148	203	201·71
1	1192	210	209·44
2	1204	196	211·55
1	1237	210	217·01

76   Mean . 1069·4   Mean . 187·9

The probable error[1] in this series is 9·929 lbs.
The permissible error is 7 lbs.

[1] The probable error was obtained by first finding the mean of all the observations above the mean, and subtracting the mean from it ; this gives the mean error in excess. The mean fall— the observations below the mean—is then found and subtracted from the mean, which gives the error in deficiency. Adding the two together and taking half gives the mean error, and two-thirds of the mean error gives the probable error.

SERIES B.

No. of Observations.	Body Weight. lbs.	Estimated Weight-carrying Power. lbs.	Calculated Weight-carrying Power. lbs.
5	1015	183·4	174·39
7	1049	187·0	180·22
8	1081·5	194·25	185·81
6	1099	195	188·82
10	1133	194·6	194·66
6	1156·17	197·17	198·55
9	1186	202·0	203·76
2	1233	217	211·84
2	1239	213·8	212·87
2	1274	215·0	218·88
1	1295	196·0	222·49
2	1333·5	222·5	229·1

60　　Mean .　1136·6　Mean .　195.28

Probable error, 6·072 lbs.
Permissible error, 7 lbs.

If we now take the totals in the two series, we find that the mean body weight of the 136 horses is 1103 lbs.; the mean carrying capacity, 191·59 lbs.; the mean ratio of carrying power to body weight, 1 : 5·757. That is to say, it takes, speaking roughly, 5¾ lbs. of body weight to carry 1 lb. on the back during severe exertion.

If we take, for convenience, the average weight of a riding horse to be 1000 lbs., the weight he is capable of carrying hunting would be 173·71 lbs., and this may certainly be regarded as a close approximator to the truth. For horses performing slower work 28 lbs. may be added to the above, making, in round numbers, 200 lbs. as the load capable of being carried by a horse of the above weight.

The rule to ascertain the carrying power of a horse is to divide his body weight by 5·757, and, if intended for only moderate work, add to this 28 lbs.

It has been suggested to me that a possible objection to my argument might be that the experts' judgment of weight-carrying power largely depends on their estimate of the horse's weight, and that what I chiefly measure may be little or no more than the accuracy of appraising weight.

I cannot, however, believe this to be the case; horses in this country are not judged by weight, the subject of body weight enters into none of the questions connected with the utilisation of horses, so that there is hardly likely to have been any bias in that direction in the minds of the experts who assisted me. The power of recognising the weight a horse should carry is a something peculiar to a few men—they cannot tell you how they know, nor can they impart their knowledge to others. All I claim here is that if we have not succeeded in learning what it is that constitutes this remarkable judgment, we have at least learned another method by which this important practical information can at once be obtained, without waiting years to acquire the needful experience and knowledge.

ON THE NATURE AND SIGNIFICANCE OF LEUCOCYTOSIS.[1]

By ROBERT MUIR, M.D., F.R.C.P., Edinburgh, Professor of Pathology, University College, Dundee.

Introductory.

THE subject of the discussion which I have the honour of opening is at once one of the most important in modern pathology, and has also a most important bearing on clinical diagnosis and prognosis. It is, moreover, one which has very wide connections, being, on the one hand, closely involved with the tissue changes usually grouped together as inflammation and, on the other hand, intimately related to the defence of the organism against noxæ of various kinds, especially against microbes. The subject is, in fact, so wide, that on such an occasion as the present it can only be treated in one or two of its parts. The object or meaning of local increase, emigration of leucocytes, and their subsequent behaviour would imply in its discussion a consideration of the whole question of phagocytosis and of the bactericidal or other antagonistic properties (direct or indirect) of the substances secreted by or contained within these cells. And further, the observations on the modification in the number of leucocytes within the blood as a whole, or the modification in the number of different varieties, as observed clinically, are now so numerous that a consideration of them would occupy much longer time than I have at my disposal. Some of these observations must be incidentally referred to, whilst other speakers may refer to them in greater detail. I shall take as the main subject of my remarks the nature of leucocytosis as a process in general pathology. What is the fundamental change on which the leucocyte excess depends, and what is its standing in relation to the question of tissue reaction?

Definition of Terms.

The term leucocytosis may be applied to any condition in which there is an excess of leucocytes over the normal, whether in the tissues or part of the vascular system, a local leucocytosis, or throughout the circulating blood, a general leucocytosis. We shall first speak of the latter. The general facts are well known—that in many acute inflammatory conditions, occurring naturally or experimentally produced, such as pneumonia, erysipelas, etc., and especially in suppurative conditions, the number of leucocytes in the circulating blood may be two- or three-fold the normal, or even more. Further, a similar condition may be produced by the introduction into the body of various chemical substances, of which the most important classes may be said to be (a) bacterial products, especially the so-called bacterial proteins; (b) extracts of various organs or tissues rich in cells, such as spleen, bone-marrow, etc.; (c) a great variety of definite organic compounds—for example, peptone, curare, nucleic acid, etc. Leucocytosis produced by such substances is sometimes spoken of as toxic leucocytosis, but in its essence is closely related to that occurring

[1] An address in the section of Pathology at the annual meeting of the British Medical Association, 1898. Reprinted from the British Medical Journal, 3rd September, 1898.

in inflammatory conditions.   One question has been raised which must first be answered—namely, is the leucocytosis occurring in the conditions mentioned real? that is, does the total number of leucocytes in the circulating blood increase, or is there simply an accumulation in the peripheral vessels from which blood is usually drawn?   Recent observations, of which those of Goldscheider and Jacob may be mentioned (with which my own, so far as they go, agree), show that the increase is a general one, and that in the peripheral vessels there is the same proportionate excess over the number in the blood of the heart as there is in the normal condition.   There occurs therefore in leucocytosis an increase of the total number of leucocytes in the circulating blood, or, in other words, an addition of leucocytes to the blood.   Further, in the case of suppurative and allied conditions, there occurs an enormous emigration of leucocytes from the vessels, so that we see, for example, in clinical cases, several ounces of pus discharged daily, and during this time, be it noted, the number in the blood is much above the normal.   In such conditions, where there is a continuous drain, and at the same time an excess of leucocytes in the blood, the production must be much in excess of the normal.   In the case of leucocytosis following injections of chemical substances there must first be an addition of cells to the blood, but after this addition has taken place it does not necessarily follow that it requires an increased multiplication to maintain it, though such may occur.

There are two points which also must be kept in view: (1) The variety of leucocyte in excess in these conditions is almost exclusively the finely granular (oxyphile or neutrophile), actively amœboid leucocyte, with polymorphous nucleus; (2) the enormous rapidity with which the leucocyte increase occurs—namely, a doubling or more of the normal number in a few hours.

Varieties of Leucocytes.—The Origin of the Finely Granular Leucocytes.

[The varieties of leucocytes, as met with in the blood of man and animals were shortly described.]   The distribution of these varieties is also of importance, but I wish specially to refer to the chief sites of multiplication of leucocytes, as this seems to me a point upon which sufficient stress has not been laid.   In the first place, as everyone knows, leucocytes multiply by mitotic division in lymphoid tissue, lymphatic glands, Malpighian patches of spleen, solitary glands, etc. The cells there undergoing division are small spherical cells, with relatively large nucleus and rather scanty hyaline protoplasm. These cells give rise to lymphocytes, which leave by efferent paths, and a certain proportion reach the blood by the thoracic duct. Secondly, leucocytes undergo division in the bone marrow.   The great proportion of cells dividing there are of somewhat larger size than in the germ centres, and their protoplasm contains fine granules which have a definite (oxyphile, neutrophile, etc.) reaction.   The coarsely granular eosinophile cells also multiply in the same position, though they are fewer in number, and their multiplication is less active, than is the case with the former variety.   Now it is to be noted that division among the finely granular cells in the marrow in normal conditions is very active; in fact, so far as I have seen, as active

as in the germ centres of lymphoid tissue. How do the leucocytes derived from these cells leave the marrow? One naturally concludes by the blood stream, but in the blood stream there are no cells with spherical or oval nucleus and with fine granules in the protoplasm, which have the same reaction. Examination of the marrow shows, however, that the cells there present show great variation in the configuration of the nucleus, and, in fact, one can trace all the intermediate stages from the simple rounded or oval nucleus through various degrees of lobulation to the markedly multipartite nucleus of the finely granular, actively amœboid leucocyte. And, if one examines sections, one can see that leucocytes of this character are normally found in varying number at the periphery of the blood channels, whilst the larger cells from which these cells, in my opinion, spring, are, as a rule, further removed from the blood stream. According to this view, then, the finely granular cells after division undergo this peculiar change in the nucleus which specially suits them for amœboid movements, and enter the blood in their fully formed condition. I may add that I have failed to find any amœboid movements in the larger cells in the marrow. The acquisition of this property would appear to be developed *pari passu* with the change in the configuration of the nucleus.

Another fundamental question in the inquiry is whether any formation of the finely granular leucocytes with polymorphous nucleus takes place in the blood from the larger hyaline forms with simple spherical nucleus. There is no doubt that in the latter, indentation and a certain amount of lobulation of the nucleus may be seen ; but does the change go further? It will be found that authors disagree markedly on this point. At one time I considered that I could find in human blood transition forms between the "uninucleated" and "multinucleated" leucocytes, as they were formerly called, but such transition forms appear to be exceedingly scanty and apparently quite insufficient in number in leucocytosis to account for the large number of multinucleated forms. If we investigate the question in the blood of the rabbit in which the corresponding cells have the granules much more pronounced, I think it will be impossible to observe transition forms ; in fact, the whole appearance during the occurrence of a leucocytosis is as if a large number of cells of exactly the same kind were rapidly added to the blood. On carefully considering, on the one hand, the character of the cells in the blood, and, on the other hand, the cells in the marrow, there can be little doubt—in my mind there is no doubt—that the polymorphonuclear leucocytes are formed chiefly, if not entirely, from the latter, and enter the blood stream in their fully formed condition.

Leucocytosis in Inflammatory Conditions.

We must now pass on to the consideration of the evidence of increased multiplication of leucocytes in inflammatory conditions associated with leucocytosis. I may state, in a word, that neither in the tissues nor in the blood stream is there evidence of multiplication by mitosis amongst the polymorphonuclear leucocytes to account for the increase. The question regarding direct division is a difficult one, but here again facts are entirely wanting to show that such a process plays an important part in the changes which occur. If these

conclusions are justified, an important question arises—namely, Are any important changes found in the bone-marrow when there is a constant drain on this variety of cell; in other words, when there is an increased production of this form of cell? To answer this question I have made a considerable number of experiments, and shall briefly detail the results. These experiments consisted in producing leucocytosis, or at least a removal of leucocytes from the blood, by subcutaneous or intraperitoneal injections of organisms, chiefly staphylococci. The experiments lasted for various periods of time up to four weeks and a half. The results are somewhat striking. I find that there occurs an important change which is readily recognisable. This change consists in a general absorption of the fat of the marrow and a corresponding hyperplasia of cells; and, further, the important point is, that the cells increased in number are the large finely granular cells which may be called finely granular leucoblasts, and which are the source of the finely granular leucocytes. The number of mitotic figures present shows that these cells are undergoing rapid multiplication. The coarsely granular eosinophile cells are relatively, and it may be absolutely, diminished. Erythroblasts or nucleated red corpuscles are also much diminished. The giant cells may undergo degenerative change and in great part disappear. (These cells do not contain granules, and are probably derived from the larger hyaline cells.) The marrow in such a condition presents a striking deviation from normal, and the change may be said to be of a corresponding nature to what occurs in the erythroblasts after hæmorrhage (great increase in their number and increase in the mitotic figures). If we call the latter the "erythroblastic" type of marrow, then we may call that which I have described the "leucoblastic" type of marrow. This fact appears to me to throw light on the whole subject. If we consider the finely granular cells as a class—say in a case of suppuration—we find (*a*) locally an enormous number of finely granular leucocytes (plus corpuscles); (*b*) in the blood a great increase in the number of the same cells; and (*c*) in the marrow a great increase in the cells from which these leucocytes are derived.

Furthermore, it is very probable that the same cause would explain all these phenomena. It must now be accepted that bacterial products act as chemotactic substances, and are the chief agents in the leucocyte emigration and accumulation. One cannot, I think, study the phenomena which follow, for example, intraperitoneal injection of various bacteria, without recognising that this process must be at work. Granted then a chemotactic substance, it may be in such quantity as only to act locally—in other words, we may have a small local inflammation or suppuration without leucocytosis, but if the local lesion be more extensive, then these substances which produce chemotaxis are absorbed by the blood stream in such quantities as to act on the leucocytes lying at the periphery of the blood stream in the bone-marrow, and the result will be a general leucocytosis. This is in all probability the first factor in the production of the leucocytosis quickly following the injection of chemical substances into the circulation. But there is also the increased multiplication of finely granular leucoblasts (marrow cells), and this is probably due to a stimulating effect which these substances exert on the cells—it cer-

tainly cannot be explained on any mechanical principle. We have, therefore, the local leucocytosis and the general leucocytosis induced by the same agency. This view very closely corresponds with what Ehrlich and Lazarus have adopted in their latest publication.[1] I may say, however, that I have arrived at these conclusions quite independently, and that, so far as I know, the changes referred to in the bone-marrow have not been previously described.

In interpreting the phenomena we may then say: (1) That the local leucocytosis is a most important means of defence; (2) that the proliferative changes in the bone-marrow are the means by which the leucocytes concerned may be supplied in large numbers at any given place of need; (3) that the leucocytosis in the blood is an indication at least that this supply is being maintained. We may also mention that the vascular arrangements in the marrow are such as, on the one hand, to permit a ready action upon its cells of chemotactic substances circulating in the blood, and, on the other hand, to allow a free and rapid passage of the cells into the blood.

It is to be noted that chemical substances must be the means by which a general leucocytosis is brought about, and, therefore, it is not surprising that it can be produced where there is no local inflammatory change. Whether in such a case the leucocytosis really acts as a means of defence by combining with toxins in the blood, for example, in the leucocytosis following injection of diphtheria toxin, must still be considered an open question. Another question worthy of study is whether, and if so to what extent, degenerating or broken down tissues act as chemotactic agents in the production of a general leucocytosis.

Relations of the Finely Granular and Hyaline Leucocytes.

I have said that in view of their chief sites of multiplication the great majority of leucocytes fall into two series, one including the lymphocytes and hyaline cells, the other including the finely granular leucocytes and the finely granular leucoblasts of the bone-marrow, with possibly the eosinophile series. (The consideration of the relation of basophiles has necessarily been omitted; they certainly play a minor part in the questions under consideration.) An important question, and one exceedingly difficult to answer, is whether or not hyaline cells acquire granules as they lie in the bone marrow and become finely granular leucoblasts. This is a question which I am not prepared to answer, either in the affirmative or in the negative, but one must note, first, that during a prolonged leucocytosis mitosis in the finely granular leucoblasts is sufficient to account for the leucocyte increase; and secondly, that although hyaline cells are normally present in the bone marrow, these do not become increased during leucocytosis. Whilst, then, the transformation is quite possible, I have not found sufficient evidence from my experiments that it plays an important part in the phenomenon of leucocytosis.

Another point of importance may be referred to, namely, that if we consider the two series of leucocytes as above described, and suppose an excessive proliferation of one or other, we have an explanation of the two forms of leucocythæmia. In the one form, as is well known, there is an increase in the blood and tissues of the lymphocytes, and

[1] Die Anämie, in Nothnagel's "Specielle Pathologie und Therapie."

it may be of the larger hyaline cells.   In the other form we have an increase of the finely granular leucoblasts (marrow cells) and an appearance of them in the blood, and at the same time a great increase in the number of finely granular polymorphonuclear leucocytes.

Leucopenia.

A few words must also be said with regard to diminution in the number of leucocytes—leucopenia—and we may consider, first, the fall which may occur with comparative rapidity in inflammatory or infective conditions, or on the introduction of certain chemical substances into the blood.   Here, it is to be noted, the fall is chiefly, sometimes almost entirely, on the part of the finely granular polymorphonuclear leucocytes.   One may summarise thus the circumstances on which this fall may depend :—

First, the rapid fall after the injection of various chemical bodies is due in great part if not entirely, to the accumulation of leucocytes in the capillaries of various organs, especially of the lungs.   This has been proved by the experiments of Surgeon-Captain D. Bruce, of Goldscheider and Jacob, as well as my own.   It can readily be demonstrated.   Therefore one may say that the rapid destruction of leucocytes supposed by Löwit to occur in these circumstances has at least not been proved.

Secondly, after injections—for example, of organisms into the peritoneum—the fall may be due in part to accumulation of the leucocytes in the capillaries of the mesentery, peritoneum, etc., and in part to the emigration of leucocytes into the peritoneal cavity, the latter occurring very rapidly.   It is, therefore, possible that a sudden extension of the inflammatory lesion may produce a fall in the number of leucocytes.   (Where, however, the inflammatory change is more gradually developed, as in most clinical cases, leucocytosis may not be preceded by leucopenia.)

Thirdly, in severe septicæmic and toxic conditions an enormous leucocyte degeneration and destruction can be traced in the spleen, and to a less extent in the bone-marrow, and in certain circumstances there is great leucocyte accumulation associated with phagocytosis in these situations.   The removal of leucocytes from the blood stream by their being entangled in coagula forming in the heart before death —for example, in pneumonia—may also be mentioned.

Fourthly, the fall may be due to interference with the proliferative changes in the bone-marrow, as a result of the action of toxic substances.   On this point further observation is necessary.

Fifthly, whether substances exert a negative chemotaxis on the leucocytes in the marrow or elsewhere, and interfere with their passage into the blood stream, must still be left an open question.

It will be observed that in many of the circumstances mentioned the conditions determining the leucopenia are of an unfavourable kind, whether they be excess of toxic substances in the blood, excess of leucocyte emigration due to rapid inflammatory spread, rapid leucocyte destruction, etc.   No doubt in most cases a combination of circumstances is at work—for example, in the fall of the leucocytes in pneumonia without a true crisis.   But it is extremely important that in all conditions where there is leucopenia the circumstances

which bring it about should be accurately ascertained, not simply to establish scientific facts, but also to afford the basis of possible therapeutical measures.

Another interesting fact may be noted, namely, that in various grave inflammatory or infective conditions when the leucocyte number falls, there appear in the blood stream some of the finely granular leucoblasts of the marrow—the myelocytes which are so numerous in the blood in medullary leucocythæmia. Whether this is due to organic change in the marrow interfering with the normal relation of its cells I at present cannot say. It, however, appears to be usually a bad omen.

Lastly, we may merely mention that the leucocyte number is diminished in several chronic conditions—for example, in pernicious anæmia, in some cases of chlorosis (as I pointed out several years ago), and in some cases of anæmia with splenic enlargement. Here, again, the diminution is chiefly on the part of the polymorphonuclear leucocytes ; and this association with defective blood formation, or with alterations in the function of the bone-marrow, is worthy of note.

Eosinophile Leucocytosis.

Though leucocyte increase is usually due to increase in the finely granular leucocytes, attention has been, within late years, drawn to the conditions in which eosinophile leucocytes are proportionately in excess, though the total number of leucocytes may be little increased —the condition known as "eosinophilia." Local accumulation of the eosinophile cells is common in a variety of conditions. I have observed it in various abnormal conditions of epithelium, for example, in atrophic conditions of the gastric and other mucous membranes, around cancerous growths, especially some varieties of epithelioma ; in some forms of malignant glandular affection allied to the lympha-denoma ; also in some subacute inflammatory conditions, for example, in organising serous inflammations, and even in the wall of abscesses. In various affections of the skin, pemphigus, psoriasis, and several others, the number of leucocytes in the tissues may be very great. And further, it has been observed that in bronchial asthma the leucocytes in the bronchial secretion are chiefly eosinophiles. The discussion of the behaviour of these cells would occupy much too long, but it is interesting to note that a close analogy can be drawn between this form of leucocytosis and the ordinary form. There may be local accumulations of the eosinophile leucocytes without increase in the blood ; but, on the other hand, if the area affected, for example a skin lesion, be extensive, there may be an eosinophile leucocytosis in the blood. It is to be observed also that large eosinophile cells with simple nucleus are normally present in the marrow, and that mitotic figures may sometimes be observed in them. So also in the medullary form of leucocythemia these cells, both the eosinophile marrow cells and the smaller eosinophile leucocytes, may be impli-cated in the leucocyte increase. Important questions in connection with this subject are: Do finely granular leucoblasts ever become eosinophile leucoblasts, and do the finely granular leucocytes in the blood or in the tissues become eosinophile leucocytes? Some facts suggest that these two varieties of granular cells are closely related to one another, whilst others show that, to a certain extent, they

behave as if they were independent. The great diminution in the number of the eosinophiles, and it may be sometimes complete disappearance, in the blood in acute inflammatory leucocytosis is also worthy of note. This subject must be investigated along the same lines as the behaviour of the finely granular cells. The occurrence of local accumulations of lymphocytes and hyaline cells can only be mentioned as a subject also worthy of consideration.

Conclusion.

In conclusion, may I express the hope that this discussion may aid in determining the position which the term inflammation is to occupy in the science of pathology. It appears that much discussion might be avoided if a sharper distinction were drawn between the active and the passive phenomena concerned. By the former, I mean those which are of the nature of a defensive reaction ; by the latter, those which are merely the evidence of the action of toxins or other noxæ on tissues, especially on living cells. The former ought to be regarded in the light of evolution as a provision for the protection of the organism, the latter merely as evidence that living structures are necessarily capable of damage.

THE INFECTIVITY OF THE BLOOD, MUSCULAR TISSUE, AND LYMPHATIC GLANDS IN GENERALISED TUBERCULOSIS OF THE OX.

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IN a former article[1] I described certain experiments in which generalised tuberculosis was experimentally induced by the injection of materials containing tubercle bacilli into the systemic venous system of cattle. In that article the results were considered only with regard to the distribution of the macroscopic and microscopic lesions induced in the experimental animals, and in summing up it was expressly stated that the apparent absence of tubercles from particular organs in cases of generalised tuberculosis was not to be taken as reliable evidence that such organs were free from tubercle bacilli and fit for human food.

The experiments in question showed that an irruption of large numbers of tubercle bacilli into the systemic venous system determines the development of an abundant crop of tubercles in the lungs, and that at a time when these tubercles have attained a distinctly macroscopic size, the spleen, liver, kidneys, and other parts of the body may appear perfectly normal to the most searching naked-eye examination, and even to a microscopic search involving the examination of a large number of sections. But while the main purpose of the experiments was to throw light on the distribution of the lesions in generalised tuberculosis, and thus to obtain a rule for guidance in the slaughter-house inspection of tuberculous cattle, the opportunity was also taken to obtain evidence regarding the infectivity of the flesh and other parts of animals in which the disease has become generalised.

It had, of course, to be recognised that even the negative result of a prolonged microscopic search for tubercles was not conclusive evidence that tubercles and tubercle bacilli were absent from any given organ, and that recourse must be had to inoculation or feeding experiments to settle the point. In the following pages an account will be given of such experiments made with the flesh, blood, etc., of four different animals. To save repetition it may be stated at the outset that in every step of these operations the precautions necessary to avoid accidental infection were taken. The hypodermic syringe and needle were invariably sterilised by boiling in water immediately before use, and that portion of the latter introduced under the skin was not touched with the fingers during the operation. The syringe employed was one of metal and glass with an asbestos piston. The skin at the seat of inoculation was washed with 2 per cent. solution of lysol in water.

First Series.

The materials used for this series of experiments were taken from a cow which was killed on the twenty-third day after the injection of 3 cc. of bouillon holding in suspension a great number of tubercle bacilli from the mesenteric gland of a horse (Experiment I. of last article). The *post-mortem* examination of the cow revealed a dense crop of macroscopic (size of mustard seeds) tubercles in the lungs, and tuberculous enlargement of the bronchial, mediastinal, and tracheal lymphatic glands. The other groups of lymphatic glands, as well as the liver, spleen, kidneys, and muscular tissue, appeared normal, but microscopic examination revealed the presence of a few minute tubercles in the liver and spleen.

Blood.—On the day following that on which the injection of tubercle bacilli into the jugular vein was made 6 cc. of blood were withdrawn from the same vessel (left jugular) by means of a sterile hypodermic syringe. Three cc. of this were injected into the peritoneum of each of two rabbits (A and B).

Rabbit A was found dead on the forty-fourth day after inoculation. The *post-mortem* examination revealed the peritoneum normal and no trace of tuberculosis anywhere. The animal was emaciated, and the intestine contained numerous coccidia.

Rabbit B was killed on the sixty-ninth day after inoculation. The *post-mortem* examination showed the peritoneum, lymphatic glands, and thoracic and abdominal organs normal in appearance.

A rabbit (C) was inoculated intraperitoneally with 10 cc., and a guinea-pig subcutaneously with 5 cc., of defibrinated blood taken from the jugular vein of the cow immediately after death (by chloroform). This was on the twenty-third day after the cow had been inoculated intravenously. The blood was taken with the strictest antiseptic precautions, the instruments and vessel having been sterilised by boiling, while the bunch of wires used in defibrination had been heated in the flame of a Bunsen burner.

Rabbit C was killed on the forty-seventh day after inoculation, and the *post-mortem* examination revealed no trace of tuberculosis in any part of its body. The above guinea-pig was killed on the 104th day after inoculation, and the *post-mortem* examination revealed the fol-

lowing.   At the seat of inoculation (between the scapulæ) there was an
ulcerating sore from which thick caseous pus was discharging.   On both
sides the axillary lymphatic glands were enlarged to the size of a
haricot bean, and caseous.   The intermaxillary and cervical glands
were also enlarged and caseous.   Numerous tubercles in the lungs and
liver and a few in the spleen.   Tubercle bacilli were easily discovered
in these lesions.

   Muscle.—5 grammes of muscular tissue taken from the internal
muscles of the thigh of the cow with sterile instruments were
pounded in a sterile mortar with 10 cc. of bouillon, and of the turbid
liquid thus obtained 1½ cc. was injected under the skin on the right
side of the abdomen in each of two guinea-pigs (A and B).

   Guinea-pig A was killed on the forty-seventh day after inoculation.
The precrural lymphatic gland on the right side was enlarged to the
size of a horse bean and softened centrally while the inguinal gland
on the same side was slightly enlarged, and a few small tubercles
were present in the spleen.

   Guinea-pig B was killed on the 105th day after inoculation.
The skin at the seat of inoculation was intact, but under it there
was a small caseous nodule.   The precrural lymphatic gland
on the same side (right) was as large as a horse bean and caseous.
The spleen and liver were much enlarged and full of tubercles.
Tubercles were more sparingly present in the lungs.

   Popliteal Lymphatic Gland.—The left popliteal lymphatic glands
of the cow was seared on its outside and then torn open with
sterile instruments.   The tissue of the interior of the gland was
then scraped with a sterile knife, and the scraping was suspended
in 10 cc. of bouillon.   Two guinea-pigs (A and B) were inoculated
subcutaneously on the abdomen with the turbid liquid, one (A)
receiving 2 cc., and the other 3 cc.

   Guinea-pig A was killed on the forty-seventh day after inoculation.
There was a caseating lesion in the texture of the abdominal wall.
The precrural and axillary lymphatic glands on the same side were
much enlarged and caseous.   The corresponding glands on the
opposite side were slightly enlarged and commencing to caseate.
The spleen contained numerous tubercles, and the liver and lungs a
few.

   Guinea-pig B was found dead on the ninety-ninth day after inocu-
lation, with an ulcerating lesion at the seat of inoculation on the
abdominal wall, the precrural glands on both sides enlarged and
caseous, and tubercles in the spleen, liver, and lungs.

   Bronchial Gland.—A guinea-pig was inoculated with a scraping
from the left bronchial lymphatic gland of the cow suspended in 1⅓ cc.
of bouillon.   It was killed on the forty-seventh day after inoculation.
There was an ulcerating sore at the seat of inoculation, the precrural
and axillary lymphatic glands on both sides were enlarged and
caseous, and numerous tubercles were present in the spleen, liver,
and lungs.

Second Series.

   The materials used for this series of experiments were furnished by
a cow (Experiment II. of last article) which was inoculated intra-
venously with 2 cc. of bouillon holding in suspension tubercle bacilli

from a tuberculous mesenteric gland of a horse. The cow was killed on the thirty-sixth day after inoculation. The *post-mortem* examination revealed a miliary tuberculosis of both lungs, and tuberculous enlargement of the bronchial and mediastinal lymphatic glands. Tubercles were also present in the pharyngeal and prescapular lymphatic glands on the left side. The liver, spleen, and kidneys appeared normal to the naked eye, and no tubercles could be found in them on microscopic examination.

Popliteal Lymphatic Gland.—This gland (the left) was treated as in the previous case, and the material scraped from its interior was suspended in bouillon, of which 5 cc. were injected under the skin of the abdomen of a rabbit and 2 cc. were similarly injected into a guinea-pig.

The rabbit was killed on the thirty-fourth day after inoculation. The *post-mortem* examination showed about a dozen caseating tubercles in the substance of the abdominal wall, the largest about the size of an oat-seed. The axillary lymphatic glands on the same side were enlarged and caseating. No tubercles were present in any of the organs of the thorax or abdomen.

The guinea-pig was also killed on the thirty-fourth day after inoculation. A caseating lesion was present in the abdominal wall at the seat of inoculation, and the precrural lymphatic glands on the same side were enlarged to the size of a horse bean and softened centrally. Numerous miliary tubercles were present in the liver, and the spleen contained one pin-head tubercle.

Spleen.—Material scraped from the depth of the spleen pulp was suspended in bouillon, of which 4 cc. were used to inoculate a rabbit and 1 cc. a guinea-pig, each subcutaneously on the abdomen.

The guinea-pig was found dead on the eighteenth day after inoculation. No lesion was discoverable in the abdominal wall, but numerous pin-head tubercles were present in the liver and spleen. No tubercle bacilli could be found in them. A fragment of liver containing a tubercle was introduced under the skin of the abdomen of a rabbit. This rabbit was killed seventeen months afterwards and found to be healthy.

The rabbit was killed on the thirty-fifth day after inoculation. In the abdominal wall behind the axilla and a little in front of the seat of inoculation there was a caseating nodule smaller than a pea. An axillary lymphatic gland on the same side was enlarged to the size of a garden bean, but not visibly degenerated. The abdominal and thoracic viscera were normal.

Muscle.—Material scraped from one of the muscles in front of the thigh was suspended in bouillon, and 4 cc. of the liquid were injected under the skin of the abdomen of a rabbit, and 1 cc. at the same place in a guinea-pig. A kitten was fed for about a week with portions of the same muscular mass, and during that time it consumed 22¾ ounces.

The rabbit was killed on the twenty-sixth day after inoculation. On the outer side of the left fore-arm the skin was denuded of hair and ulcerating. A similar condition of skin existed on the right side behind the elbow. On reflection of the skin at these places the subcutaneous tissue was found to be much thickened, largely necrotic, and broken down to a thick yellow purulent material. A similar

lesion was present in front of the right shoulder. The axillary glands were swollen and juicy. Cover-glass preparations from these lesions showed some filaments like those of the necrosis bacillus and abundant short ovoid bacteria. No tubercle bacilli. Abdominal and thoracic viscera normal.

The guinea-pig was found dead[1] 183 days after inoculation. It showed no tuberculous lesions.

The above kitten was killed on the seventy-fourth day after the commencement of the feeding experiment. It showed no tuberculous lesions on *post-mortem* examination.

Lung.—A scraping from the diseased lung tissue was suspended in bouillon, of which 1½ cc. was injected under the skin of the abdomen of a guinea-pig (A), and ½ cc. at the same place in another guinea-pig (B).

Both guinea-pigs were killed on the thirty-fifth day after inoculation, and the lesions discovered at the *post-mortem* examination were almost identical in the two cases. There was an ulcerating sore at the seat of inoculation, and on the same side the precrural, inguinal, and axillary lymphatic glands were enlarged and caseating. On the opposite side the precrural and axillary glands were in a similar condition, and the inguinal group was enlarged but not visibly caseous. Tubercles were numerously present in the spleen, liver, and lungs.

Third Series.

The parts tested for experiment in this series were taken from a steer (Experiment III. of last article) which was killed on the thirty-seventh day after intravenous inoculation with a scraping from the tuberculous lung of a cow. The *post-mortem* examination revealed a dense miliary tuberculosis of both lungs, with tubercles in the enlarged bronchial and mediastinal lymphatic glands. The other lymphatic glands of the body and the liver, spleen, and kidneys appeared free from tubercles on naked-eye examination. Microscopic examination revealed the presence of a few tubercles in the liver, but none in the spleen or kidneys.

Blood.—At the time of slaughter blood was taken in a sterile vessel and defibrinated by whisking with a coil of sterile wire. Of this defibrinated blood 5 cc. were injected under the skin of the abdomen of a rabbit, and 2 cc. at the same place in a guinea-pig.

The guinea-pig was killed on the 119th day after inoculation. No tuberculous lesions were present at the seat of inoculation or elsewhere.

The rabbit was killed on the 126th day after inoculation. The seat of inoculation, the lymphatic glands of the body, and the abdominal organs were normal. A portion of each lung towards its apex was solid from catarrhal pneumonia. No tubercle bacilli could be found in these lesions, but cover-glass preparations showed numerous small bacteria of the fowl-cholera type.

Muscle.—A portion of muscle from the inside of the thigh was seared with a red-hot spatula, and an incision into it was then made with a sterile knife. The fresh surface thus exposed was scraped with a sterile knife, and the scraping was suspended in bouillon. Three cc.

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[1] On the same day several other guinea-pigs in different cages and the subjects of different experiments were found dead, in consequence, apparently, of an error in feeding.

of this liquid were injected under the skin of the abdomen of a rabbit, and 2 cc. at the same place in a guinea-pig. The liquid contained many coarse particles, and had to be injected through a wide hypodermic needle.

Both the rabbit and the guinea-pig were killed on the 119th day after inoculation, and found to be free from tuberculous lesions.

Spleen.—The method of the last-mentioned experiment was repeated with the spleen. Both rabbit and guinea-pig were found to be healthy when killed on the 119th day after inoculation.

Kidney.—A scraping from one of the kidneys, taken as above, was suspended in bouillon, of which 2 cc. were injected into each of two guinea-pigs (A and B). Guinea-pig A was found dead on the 103rd day after inoculation. No tuberculous lesions were found at the *post-mortem*, and the cause of death was not ascertained. The animal appeared to have had diarrhœa.

Guinea-pig B was killed on the 119th day after inoculation. No tuberculous lesions were found in it.

Liver.—A scraping from the tissue of the liver was suspended in bouillon, of which 2 cc. were injected under the skin of the abdomen of a guinea-pig. The guinea-pig was killed on the 119th day after inoculation. The skin at the seat of inoculation was intact, but under it there was a partly caseous nodule as large as a horse-bean, and a pea-sized nodule of the same character was present behind the elbow joint. On the same side the precrural glands were normal, and the inguinal group were as large as an oat-seed but not visibly caseating. Tubercles were present in the spleen, liver, and lungs.

Prescapular Lymphatic Gland.—A guinea-pig was inoculated under the skin of the abdomen with 2 cc. of bouillon, holding in suspension a scraping from the centre of the right prescapular lymphatic gland. The guinea-pig was found to be healthy when killed on the 119th day after inoculation.

Popliteal Lymphatic Gland.—Material, taken as above, from the right gland was used to inoculate a guinea-pig. The guinea-pig was found dead on the ninety-fourth day after inoculation. The skin at the seat of inoculation was intact, but in the texture of the abdominal wall at that place there was a caseous nodule as large as a horse-bean. The precrural and inguinal glands on the same side were pea-sized and caseous. The inguinal gland on the opposite side was as large as a barley grain and caseous. The spleen and liver were crammed with tubercles, and a considerable number were present in the lungs.

Bronchial Lymphatic Gland. — A guinea-pig inoculated with a scraping from this gland was found dead on the eightieth day afterwards. It had an ulcerating tuberculous lesion at the seat of inoculation, enlargement and caseation of the lymphatic glands on both sides of the body, and tubercles in the liver, spleen, and lungs.

Lung.—A guinea-pig inoculated under the skin of the abdomen with a scraping from the diseased lung tissue was killed when obviously ill on the eightieth day of the experiment. It had lesions almost identical with those present in the last-mentioned animal.

Fourth Series.

In this series the experiments were made with the spleen and muscular tissue of a case of natural generalised tuberculosis in a cow

X

(Case VI. of last article, pp. 232 and 248). In addition to extensive localised lesions (of lymphatic origin), this animal had a miliary tuberculosis of the spongy portions of both lungs. The kidneys, spleen, and liver were normal to the naked eye, but microscopic examination revealed the presence of a few minute tubercles in each of these organs.[1]

Muscle.—A scraping from the muscles in front of the thigh was suspended in bouillon, of which 4 cc. were injected under the skin of the abdomen of each of two rabbits.

The rabbits were killed sixty-one days afterwards, and both were found to be healthy.

Spleen.—A quantity of spleen pulp was mixed with bouillon, and 3 cc. of the mixture were injected under the skin of the abdomen of each of two rabbits.

Both rabbits were killed on the sixtieth day after inoculation, and the *post-mortem* examination revealed almost identical alterations in each—viz., a caseating but not ulcerated lesion at the seat of inoculation, enlargement of the precrural and axillary glands on the same side, and tubercles in the spleen, kidneys, and lungs.

Reviewing the whole of the preceding experiments, it will be seen that three of them were made with blood, and that one of these yielded a positive result. In that case the defibrinated blood was taken from a cow twenty-three days after the intravenous injection of tubercle bacilli. The guinea-pig which developed the tuberculous lesions had been inoculated subcutaneously with 5 cc. of blood ; but a rabbit which received double that quantity into the peritoneum remained healthy. A further notable point is that the blood of the same cow, when tested in quantities of 3 cc. on the day after the intravenous injection of bacilli, yielded negative results. Probably the explanation of these apparently discordant results is to be sought in the following considerations. At the time when the cow was killed the bronchial and mediastinal glands were enormously enlarged and the seat of very active disease, and it is not improbable that bacilli had already begun to leave these glands by way of their efferent lymphatic vessels. In other words, a process of natural generalisation of the disease had already begun, and occasional bacilli were finding their way into the blood stream by way of the thoracic duct. The fact that the rabbit escaped while the guinea-pig became infected indicates that only a very small number of irregularly distributed bacilli were present in the blood.

The muscular tissue was tested in each of the four cases, and in only one of them were the results of the experiment positive.

The spleen pulp was tested in three of the cases, and in two of these the result was positive. In one of the positive cases subsequent microscopic examination showed some minute tubercles in the spleen pulp, while in the other positive case and in the negative case no tubercles could be found.

An apparently healthy popliteal lymphatic gland was tested in three of the four cases, and in every instance the result was positive in each of the inoculated animals. In one of these three cases an

[1] These were not detected until after the inoculations had been made.

experiment made with a prescapular lymphatic gland had a negative result.

The kidney was tested in one of the cases and the liver in the same case. The result was negative in the two animals inoculated from the kidney, and positive in the one animal inoculated from the liver. Subsequent microscopic examination showed a few tubercles in the liver and none in the kidney.

The experiments made with the visibly diseased tissue of the lungs and bronchial glands need not be further referred to. These were carried out in case the inoculation tests with the apparently healthy parts should all have negative results; in which case it might have been said, though not with any probability, that the lesions in the lungs were caused by, and contained only, the dead bacilli injected into the veins.

The results of these experiments serve to give fresh emphasis to the necessity for a systematic scientific inspection of animals slaughtered for food. In the absence of the lungs and thoracic glands, two of the four carcases experimented with might have been sold anywhere as sound and healthy, and yet, as the experiments proved, living virulent tubercle bacilli were diffused throughout them.

A REPORT UPON ONE HUNDRED CONSECUTIVE CASES OF MEDIAN NEURECTOMY.

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IN the *Journal of Comparative Pathology and Therapeutics* for September 1896 and September 1897 there were published accounts of eighty-seven individual consecutive animals upon which median neurectomy had been performed, ten of them being operated upon on both fore legs; since then I have in the Clinique, or after consultation with various practitioners, operated more than one hundred times, making a total of rather more than 200 neurectomies of the median nerve.

Of these I purpose tracing up from time to time the first hundred consecutive cases, in order to demonstrate the possibilities and utility of the operation, and, perhaps more particularly, the percentage of permanent successes, together with the prospects of unfavourable sequelæ.

The animals, except where otherwise stated, have been traced up to the end of September of this year. In no instance up to the present have I been able to trace a ruptured tendon or sloughing of the hoof, unless the external plantar had also been divided; and the result of a full three years' experience (the first case was operated upon on the 5th October 1895) is to confirm the remarks made in the last report, viz. :—" That for old-standing lameness, where due to splints, exostoses on the inside of the leg, chronically sprained, thickened, and painful perforans or perforatus tendons, or cases of that kind which cause pain by pressing on the adjacent nerve structures, after

all other known method of treatment has failed, median neurectomy is the operation which will give the animal a new lease of life and usefulness."

Attention was drawn in the 1897 article to a neuroma which was found in Case 24, as M. Pellerin in his pamphlet makes an especial note of its rarity, but we have several times had evidences of their presence (Cases 47, 50, and 98), and of lameness produced by them.

Referring to the cases recorded in the Journal for 1896, the terminations of Cases 1, 5, 6, 7, 10, 17, 23, 25, and 27 have already been mentioned, and in the Journal for September 1897 the terminations of Cases 2, 3, 22, 24, 28, 30, 34, 35, 37, 38, 40, 41, 42, 43, 44, 49, 51, 52, 53, 55, 60, 61, 63, 65, 69, 70, 73, 75, 76, and 79 are shown. The remainder are here traced as far as possible :—

CASE 4.—A high-stepping pony, six years old when operated upon on the 16th of November 1895, is still working regularly and well, and has not been lame since.   The animal was in the Clinique on the 8th of December with a wound on the tongue ; there is no alteration in action, the pony stepping as well as ever, and there has been no lameness in that leg since.

CASE 8.—A carriage mare, about nine years old when operated upon on the 3rd of February 1896, has not suffered from lameness in that leg since, and is at present working regularly in a brougham.

CASE 9.—An aged cab mare, operated upon on the 5th of February 1896, worked regularly in a cab without lameness until July 1898, when the animal was sold in a repository and lost sight of.

CASE 11.—A cab gelding, nine or ten years old when operated upon on the 29th of February 1896, worked satisfactorily without lameness on that leg until June 1898, when the animal was sold and lost sight of.   Owing to lameness due to severe chronic sprain of the tendons, and ringbone on the opposite leg, both median and external plantar neurectomies were performed, the former in November 1897 (causing amelioration of the symptoms), and the latter in February 1898, the effect being to enable the animal to continue in regular work until sold in June.

CASE 12.—A cart mare, six years old when operated upon on the 16th of March 1896, has since developed a very large ringbone and exostosis on the inside of the fetlock.   This caused occasional lameness after hard work during June, July, and August, and external plantar neurectomy was advised ; in the latter month, however, the animal seemed much better, and as the lameness disappeared the operation was not performed.   In October last the owner informed me that there had been no return of the lameness, and that the animal was working regularly and well.

CASE 13.—An aged van gelding, operated upon 18th March 1896, was working regularly in September 1897, but, owing to the owner having changed his address, I have been unable to trace the animal further.

CASE 14.—An aged van gelding, operated upon on the 2nd of April 1896 in both fore legs, has been working regularly and satisfactorily ever since.

CASE 15.—A very old pony, operated upon on the 2nd of April 1896, was working regularly in September 1897, but has since then been sold, and I have been unable to trace it further.

CASE 16.—An aged cab mare, operated upon in both fore legs 20th April 1896, has worked regularly without lameness ever since.

CASE 18.—An aged cab mare, operated upon 20th April 1896, was sold by its owner, and was last seen by him in June 1898, when it was working satisfactorily and free from lameness.

CASE 19.—An aged cab mare, operated upon on the 27th of April 1896, came to the Clinique in November 1897 suffering from lameness on the other fore leg, this being due to ringbone and exostosis of the fetlock; actual cautery was applied without success, and on 3rd January 1898 median neurectomy was performed. This gave immediate relief, the lameness being so slight as to be perceived only by an expert. The wound was treated in the usual manner, and the patient made an excellent recovery. Since then she has worked regularly and well.

CASE 20.—An aged cart gelding, operated upon on the 29th of April 1896, is still working regularly and well.

CASE 21.—An aged cab gelding, operated upon on the 29th of April 1896, was working regularly in September 1897, but since then I have been unable to trace it further.

CASE 26.—A van mare, nine or ten years old when operated upon on the 22nd of June 1896, showed signs of lameness again on the same leg at the end of November 1897; this gradually increased to such an extent that the animal could not continue at work, and the owner brought it to the Clinique on 12th January 1898 for further advice. Pressure over the seat of operation gave intense pain, and a neuroma as large as a filbert could be distinctly felt. This was dissected out carefully, and the lameness almost entirely disappeared when the animal was trotted immediately afterwards. The mare went to work on the 3rd of February, but fell slightly lame again early in May. A cantharides blister was applied to a little thickening which was present over the seat of operation, and the animal has been working regularly without lameness ever since.

CASE 29.—An aged cab gelding, operated upon on the 17th of July 1896, was working regularly in September 1897, but has since then been sold to a dealer and lost sight of.

CASE 31.—A cart gelding, operated upon (when seven years old) on the 29th of July 1896, has shown lameness once or twice since on the same leg, but for the past twelve months has worked regularly and satisfactorily.

CASE 32.—A cart mare, operated upon (when eight or nine years old) on the 10th of August 1896, became improved, but not fit for trotting work, directly after the operation. As this was surmised to be due to a ringbone, external plantar neurectomy was advised, but not resorted to, and in a few weeks the lameness passed off. In my last report I was unable to trace the result, but since then I have found out that the horse worked satisfactorily until about November 1897, when it was sold and lost sight of.

CASE 33.—An aged trap gelding, which had median neurectomy performed on the 13th August 1896, and external plantar neurectomy on the 28th, worked regularly at slow work until October 1897, when it was sold and lost sight of.

CASE 36.—An aged cab mare, operated upon on the 26th of August

1896, worked regularly in an omnibus until May 1898, when the animal was put in a repository and sold. Since then I have been unable to trace it.

CASE 39.—An aged cab mare, operated upon on the 18th of November 1896, is still (November 1898) working regularly and without return of the lameness.

CASE 45.—A cab horse, which had had plantar neurectomy performed on the 26th of October 1896, and median neurectomy on the near fore leg 21st December, worked regularly until June 1898, when it met with an accident, fracturing the ilium, and was destroyed. This animal had median neurectomy performed on the off fore leg on the 15th of April 1897 (*see* Case 71).

CASE 46.—A cab gelding, aged, operated upon on the 13th of January 1897, worked satisfactorily and without return of the lameness until January 1898, when it was sold and lost sight of.

CASE 47.—An aged pony, operated upon on both fore legs on the 13th and 14th of January 1897, is still working satisfactorily. There is no lameness, but a distinct neuroma can be felt at each seat of operation (September 1898).

CASE 48.—An aged pony, operated upon on the 18th of January 1897, is still working satisfactorily.

CASE 50.—A cab mare, seven or eight years old when operated upon on the 19th of January 1897. This animal fell lame again in July 1898, and a neuroma about the size of a large walnut was removed from the end of the nerve; after this the lameness gradually disappeared, and the animal is now working satisfactorily.

CASE 54.—An aged cab mare, operated upon on the 5th of February 1897, is still working regularly.

CASE 56.—An aged cab mare, operated upon on the 10th of February 1897, was working regularly in June 1898, but has now been sold and lost sight of.

CASE 57.—An aged cab mare, operated upon on both fore legs on the 18th of February 1897, is still working regularly, but shows lameness occasionally after an unusually hard day's work.

CASE 58.—A cab gelding, six years old. Median neurectomy was performed on the 18th of February 1897 and external plantar neurectomy on the 9th of March. The animal worked for twelve days, but fell lame again, and the owner decided to have it destroyed.

CASE 59.—An aged cab mare, operated upon on the 19th of February 1897, was working regularly in September 1897, but since then I have been unable to trace it.

CASE 62.—An aged cab mare, operated upon on the 11th of March 1897, worked regularly, with the exception of occasional lameness after a severe trot, until October 1897, when the animal was sold and lost sight of.

CASE 64.—An aged cab mare, operated upon on the 12th of March 1897, on the near fore leg, and on the off fore on the 17th December 1897, is still working regularly.

CASE 66.—A light van gelding, eight years old, operated upon on the 2nd of March 1897, has worked regularly ever since.

CASE 67.—A hunter gelding, nine years old, which had median neurectomy performed on the 20th of March 1897 and external plantar neurectomy on the 26th of June, was used as a hunter and

cavalry charger until November 1897, when gelatinous degeneration of the hoof took place, and the animal was destroyed.

CASE 68.—A light cart mare, aged, operated upon on the 3rd of April 1897, was working regularly in September of that year, but has since been sold, and I have been unable to trace it further.

CASE 71.—A cab gelding, suffering from lameness due to neuromata at the seat of operation of a double plantar neurectomy, and which had median neurectomy performed on the off fore leg on the 15th of April 1897, worked regularly until June 1898, when it met with an accident, fracturing the ilium, and was destroyed. This is the same horse as Case 45.

CASE 72.—A trap mare, about nine years old when operated upon on the 22nd of April 1897, with the exception of a short rest owing to occasional lameness within the first few months, has worked regularly ever since.

CASE 74.—Van mare, aged, operated upon on the 3rd of May 1897, has worked regularly ever since.

CASE 77.—Cab mare, aged, operated upon on the 17th of May 1897. This animal was working regularly in September 1897, but has since been sold and lost sight of; it had no return of the lameness whilst in the original owner's possession.

CASE 78.—Cab mare, nine or ten years old, operated upon on the 18th of May 1897. This animal worked regularly without lameness until May 1898, when it was sold owing to pecuniary difficulties of the owner, and lost trace of.

CASE 80.—A pony, aged, upon which median and external plantar neurectomies were performed on the 19th of July 1897. The animal was working regularly in September 1897, but has been sold and lost sight of.

CASE 81.—Cart mare, six or seven years old when operated upon on the 4th of August 1897, was sufficiently improved to be fit for walking work in September 1897, but has been sold and lost sight of.

CASE 82.—Pony mare, operated upon on the 15th of July 1897, was working regularly in September 1897, but has since been sold and lost sight of.

CASE 83.—Cab gelding, seven years old when operated upon on the 22nd of July 1897, worked regularly until about May 1898, when it died of some internal complaint.

CASE 84.—Cab mare, aged, operated upon on the 6th of August 1897, was working regularly in September 1897, but since then I have been unable to trace its progress.

CASE 85.—Cab mare, six or seven years old when operated upon on the 9th of August 1897, was sold by the owner to go into the country, where she is still working regularly and has been hunted several times.

CASE 86.—Cart mare, aged, operated upon on the 3rd of September 1897, became free from lameness, but, on account of a severe attack of lymphangitis shortly afterwards, was sold by the owner and lost sight of.

CASE 87.—Cab gelding, nine years old, operated upon on the 18th of September 1897. This animal worked satisfactorily until January 1898, when it was sold and lost sight of.

More complete details in connection with the above eighty-seven

cases are given in the *Journal of Comparative Pathology and Thera-peutics* for September 1896 and September 1897. The following have been operated upon since :—

CASE 88.—Chestnut light van mare, aged, which had been in the owner's possession a month, and had been lame all the time, some-times on one leg and sometimes on the other. Each leg had exostoses on the fetlock, a chain of splints, and ringbones (the latter had been severely fired).

28th September 1897. Median neurectomy both fore legs. There was only a slight improvement after the operation, but the lameness gradually passed off, and the animal was sent to work on the 21st of October. The mare has been working regularly ever since.

CASE 89.—A trap gelding, nine years, suffering from suspected navicular disease and chronic tendinitis. Plantar neurectomy had been performed with temporary relief, but now the animal was very lame again after a day's work.

28th September 1897. Median neurectomy was performed. There was no improvement for about a fortnight, but eventually the lame-ness passed off, and the animal has been working regularly ever since, with the exception of a short rest owing to lameness on the other leg. For the relief of this lameness median neurectomy was successfully performed by the veterinary surgeon in whose practice the case occurred.

CASE 90.—An aged cab gelding, suffering from a large splint under the knee, thickened tendons, and ringbone. The animal had been lame for three months, and had been fired without any benefit.

5th October 1897. Median neurectomy was performed ; there was no immediate improvement, but as the wound healed the lameness disappeared, and the animal was sent to work on the 25th. Unfor-tunately, I have been unable to trace this case further.

CASE 91.—An aged cab gelding, suffering from a tender chain of splints and ringbones in each leg. Actual cautery (pyro-puncture) had been applied without benefit. The animal was lame on both fore legs, especially the near.

21st October 1897. Median neurectomy on both fore legs. Un-fortunately, the posterior radial artery was injured, but the wound was sutured closely, and the haemorrhage soon ceased. When trotted immediately after the operation the lameness was so slight that only an expert could possibly have detected it, and in due course the animal was put to work, working regularly ever since.

CASE 92.—A very fat, aged, skewbald pony, which had been lame for two months on both fore legs from thickened tendons and ring-bone.

25th October 1897. Median neurectomy was performed on both fore legs. The animal was taken ill on the way home and placed in the Infirmary of Mr A. Boyer, M.R.C.V.S., the symptoms being those of congestion of the lungs. Death took place on the 31st, and a *post-mortem* examination showed the lungs to be very much congested and emphysematous ; in addition there were old-standing lesions of pleurisy, and I do not think that (apart from the chloroform) the operation itself had anything to do with the cause of death.

CASE 93.—A cab mare which had been lame on and off for nine months on both fore legs, the off fore being the worst. Actual cautery

had been applied without benefit about three months before to the splints and thickened tendons on each leg.

28th October 1897. Median neurectomy on both fore legs. There was no immediate improvement, but the animal eventually became sound enough for work, and has been working regularly ever since.

CASE 94.—A trap pony, very lame from a severely sprained and thickened tendon, which was hot and very tender on the slightest pressure. There was also a ringbone, but I do not think that this was causing any of the lameness. The tendons had been repeatedly blistered without improvement.

5th November 1897. Median neurectomy was performed ; there was an immediate improvement, and on the 7th the tendons were quite cool and without the slightest tenderness on pressure.

On the 13th there was no lameness, and the animal has worked regularly ever since.

CASE 95.—Van gelding which had been suffering from lameness for about two months owing to a ringbone which had been fired twice (pyro-puncture) without improvement.

8th November 1897. Median neurectomy. The animal was sent to work on the 29th, and has worked regularly ever since.

CASE 96.—Light van mare, aged, which had been in the owner's possession about six weeks, and had been lame off and on all the time. There was a large and very tender splint on the off fore leg and a ringbone.

17th November 1897. Median neurectomy. After the operation there was no lameness. The animal was sent to work on the 2nd of December, and it has worked regularly ever since.

CASE 97.—Cab mare, six years old, which had been lame about two months. The animal had a large ringbone and thickened tendons for which the actual cautery had been applied without any benefit.

23rd November 1897. Median neurectomy. The animal was sent to work on the 6th of December, but fell lame again in January ; external plantar neurectomy was advised, but the owner decided to sell the mare. I have been unable to trace the case further.

CASE 98.—A cab gelding, lame from contracted tendons and ring-bones on both fore legs ; the near fore fetlock was knuckling over very much.

23rd November 1897. Median neurectomy on both fore legs. The animal was sent to work on the 7th of December, and has worked regularly ever since ; when in the Clinique in September last we could distinctly feel a neuroma on the end of each nerve.

CASE 99.—Cab mare, ten or twelve years old, very lame on the off fore leg from a large ringbone and sprained tendons with slight knuckling over of the fetlock joint. Actual cautery had been applied without benefit, and median neurectomy was performed (27th November 1897) as a last resource. There was no improvement after the operation.

9th December. There was an improvement, and external plantar neurectomy was performed, the result being that all lameness disappeared when walking, although it could still be perceived when trotting. The owner now (December) informs me that the animal never got fit for trotting work, and was therefore sold. Since then I have been unable to trace it further.

CASE 100.—Cab gelding, about eight years old, lame from a large exostosis inside the fetlock joint. The animal had been lame off and on for eighteen months, and had been fired (pyro-puncture) about six months ago without benefit.

27th November 1897. Median neurectomy. Unfortunately, some large artery (probably the posterior radial) was cut through, and there was some little trouble in arresting the hæmorrhage. The leg became very much swollen, and the animal dragged its toe when walking. By the 10th there was only a slight swelling of the leg, and the horse did not drag its toe. The animal was working regularly in a cab in May 1898, but since then I have been unable to trace it.

When taking into account the length of time the animals have worked satisfactorily since the operation one must not forget that by far the majority were aged animals engaged in cab work, so that their lease of life in any case would not be a very long one, and the work that they are engaged in is very trying.

It will be seen on referring to the records that one case has been in constant work for more than three years, whilst at least eleven more have been regularly working for a period of more than two years since the operation.

THE ETIOLOGY OF EQUINE INFLUENZA OR INFECTIOUS PNEUMONIA.[1]

By M. LIGNIÈRES, Paris.

PART I.

IF there is an affection whose etiology is very dubious it is pneumonia, especially that which exhibits the infectious character. Our actual knowledge on the subject is far from being precise, and it is often contradictory. I propose in the present article to notice the articles which have been published on the subject up to the present time.

To avoid unnecessary length, I shall summarise very briefly the principal researches dealing with the cause of the various pneumonias.

Although I am a convinced partisan of the rôle of external influences—notably cold, overwork, etc.—in the development of pneumonia, I shall for the moment confine myself to considering only the part played by micro-organisms.

In 1874 Friedberger pointed out the occurrence of spherical bacteria, either isolated or in chains, in the lesions of infectious pneumonia (German Brustseuche).

Siedamgrotzky found several microbes, but micrococci constantly (1882).

Dieckerhoff and Mendelsohn isolated micrococci which they were unable to differentiate from the pneumococcus of the human subject (1884). In the same year Peterlein found streptococci separated by clear zones.

In 1885 Perroncito and Brazzola also discovered cocci, most frequently streptococci.

At the same date Lustig made known his researches, in which he

employed for the first time the method of Koch. He thus isolated six microbes, and recognised the specific character of one of them—a very small bacillus stainable by Gram's method, and growing with a yellow tint on gelatine.

It is necessary, however, to come to the remarkable memoir of Dr Schütz (1887) to find established for the first time in veterinary literature an opinion that appeared to be well founded.

In the hepatised pulmonary tissue, bronchi, pleuritic liquid, and discharge, this author discovered a single micro-organism, "oval in form, comparatively small, and around which one can sometimes recognise a zone, frequently coloured, but sometimes not. These organisms occur partly isolated, partly united in pairs, sometimes combined in groups, but rarely joined to form chains. The most frequently encountered form was that of diplococci. They did not retain the colour by Gram's method of staining.

"The third day after inoculation of gelatine some small spherical white colonies make their appearance. After other three days these have grown considerably, and thus become easily recognisable. A fusion of the colonies has not been observed even when they were contiguous. Microscopic examination of preparations showed that they were entirely composed of the above-described organisms. The gelatine was not liquefied, and the growth did not extend below the point to which the medium had been punctured. They also grow in an infusion of flesh. When the bouillon culture is left at rest it deposits after twenty-four hours, at the bottom of the flask, some white flocculi, while the upper part of the liquid remains limpid.

"In bouillon it is mainly chains that form, and these are interwoven in irregular contortions.

"It does not grow on the surface of solid blood serum, but very well in the drops that collect at the bottom of the tube.

"On the surface of slanting agar the bacilli form small, grey colonies, which often require magnification to make them visible. In the drops in the tube they appear as a greyish-white deposit.

"Stab cultures in agar grow in the same manner as gelatine cultures.

"The inoculation of this microbe under the skin of mice produces almost constantly a septicæmia which is fatal in from twenty-four to forty-eight hours. At the seat of inoculation one finds a greyish tissue, moist and suppurating, while the lymphatic glands situated in the region are slightly swollen. The spleen is enlarged, tense, and bluish-red in colour. The kidneys are enlarged, greyish-brown, and opaque. The liver is hypertrophied, opaque, and infiltrated with fat. The lungs are red and moist. The blood is coagulated or still liquid.

"Rabbits inoculated in the ear die in from eight to fifteen days. They show a slight tumefaction at the point of inoculation.

"The spleen was slightly swollen, and light brown in colour. The liver was deep brown, with a shade of grey, as were also the kidneys. The lungs were reddened and filled with a frothy liquid.

"In the blood and all the organs there were many bacteria which, when stained, showed a clear circle round them. Guinea-pigs inoculated on the abdomen and fowls on the wing proved refractory."[1]

[1] Schütz. Die Ursache der Brustseuche der Pferde. Archiv für Thierheilkunde for 1887. Almost literal translation.

faction of saying that, thanks to the kindness of my *confrères*, especially those of the army, who were well placed for rendering me assistance, the subjects have been as numerous as interesting. Moreover, I have had the advantage—a very considerable one in my opinion—of having at my disposal pathological products from very various sources.

Since in this note I have in view only the bacteriological aspect of the subject, I shall refrain altogether from entering into clinical details, and shall content myself with indicating very summarily the outbreaks which have particularly interested me, and from which I have drawn the materials for my investigation.

Military School at Saint Cyr.—Between the 11th December and the 1st April 1896 forty-two patients were admitted to the infirmary for "infectious pneumonia," and of this number three died.

After a period of remission, lasting from the 1st to the 19th April 1896, and in which only five animals were attacked, a second outbreak occurred ; this lasted till the second May 1897, and during it there were ninety-eight cases and five deaths.

A notable proportion of the animals attacked were over seven years old.

2nd Hussars.—From November 1895 to January 1896 sixty horses were treated for "infectious pneumonia, probably the typhoid form," and five of these died.

13th Regiment of Artillery.—The outbreak began in January 1896 and did not end till March 1897. During this period eighty-one horses were treated for "infectious pneumonia," and nine died.

The age of the subjects was as follows :—

4 years	33
5 „	24
6 „	12
7 „	5
8 „	1
9 „	5
10 „	1
Total	81

12th Regiment of Artillery.—During the training the horses of this regiment were kept in the place occupied a few days previously by the 13th Regiment, and were thus contaminated.

From June 1896 to April 1897 eighty-three patients were treated for "infectious pneumonia," and among these there were ten deaths.

The age of the subjects was as follows :—

4 years	32
5 „	38
6 „	8
7 „	3
8 „	1
13 „	1
Total	83

Private Stable belonging to a Contractor.—I was able to follow daily an outbreak of pneumonia which prevailed in this stud from February 1896 to June of the same year.

Out of a total of seventy-four horses thirty animals were attacked in succession, and one of these died.

Their ages were as follows :—

4 years	.	.	.	5
5 „	.	.	.	6
6 „	.	.	.	2
7 „	.	.	.	4
8 „	.	.	.	5
9 „	.	.	.	2
10 „	.	.	.	1
12 „	.	.	.	1
14 „	.	.	.	1
15 „	.	.	.	1
16 „	.	.	.	1
17 „	.	.	.	1
		Total	.	30

The outbreak began with some cases of sore throat. Among eight young horses recently purchased two were on the following day (15th January 1896), recognised to be affected with strangles, and soon afterwards a number of animals had sore throat or bronchitis. Finally, on the 27th February the first case of pneumonia occurred in a fourteen-year-old horse, which died on the eleventh day. After this case twenty-nine horses, including nearly all the young animals in the stud, were admitted to the infirmary for pneumonia.

17th Regiment of Artillery.—In this regiment the outbreak was particularly violent. From October 1896 to February 1897 176 horses were treated for "typhoid pneumonia," and of this number sixteen died.

The following were their ages :—

4 years	.	.	.	40
5 „	.	.	.	44
6 „	.	.	.	43
7 „	.	.	.	21
8 „	.	.	.	13
9 „	.	.	.	4
10 „	.	.	.	3
11 „	.	.	.	4
12 „	.	.	.	3
13 „	.	.	.	1
		Total	.	176

In addition to the foregoing I have received morbid specimens from various veterinary surgeons, and I have also profited as often as possible from the cases which occurred at the Alfort School.

BACTERIOLOGY.

Following always the same method of investigation, I made a bacteriological study of the viscera and pathological products of the subjects of pneumonia as if nothing had previously been done in this direction, and I was immediately struck with the almost constant presence of a microbe which one can find in the dead subject, alike in the hepatised parts of lung, pleuritic liquid, liver, spleen, kidneys, blood, bronchial mucus, and nasal discharge.

There are some cases in which all the products of the body contain many of these microbes, and others in which they are found only in the lungs. Sometimes they occur in a state of purity in the blood, pleuritic liquid, and kidneys, but ordinarily they are associated with other bacteria.

During life puncture of the hepatised parts of the lung frequently gives positive results; more rarely they are encountered in the pleuritic liquid, and exceptionally in the blood. Out of ten attempts made with the latter liquid a culture was obtained only once.

In no case does the examination of the blood of patients, whether direct or after staining, yield a positive result.

On the dead subject the bacteria which are associated with the preceding are very various and inconstant. One frequently encounters the coli bacillus, which might be confounded with the bacillus of Friedlander described in some pneumonias of the human subject.

MORPHOLOGICAL AND BIOLOGICAL CHARACTERS OF THE STREPTOCOCCUS.

The morphological and biological characters of the microbe with the almost constant presence of which I was so soon struck, are as follows :—

It is a streptococcus formed by slightly oval elements, which divide in the direction of their transverse diameter. The long chains often appear like diplococci joined end to end.

This streptococcus is ærobic and perfectly anærobic. It is easily stained with aniline dyes, and retains the colour with the methods of Gram and Weigert.

CULTURES.[1]

Bouillon.—It grows well in bouillon, especially if that is peptonised. Generally some whitish flocculi form and fall to the bottom of the vessel, leaving the liquid very limpid. These flocculi are made up of long interwoven streptococci. Sometimes in place of the flocculi one obtains numerous granules, which are soon precipitated as a fine powder. In bouillon, notably in that from the horse, which contains a large quantity of glycogen, one observes a decidedly acid reaction.

Very good cultures are obtained in peptonised bouillon to which some serum has been added, but the medium becomes acid unless the proportion of the serum is at least equal to that of the bouillon.

The addition of glycerine does not seem to produce any modifications in the cultures.

Vegetable infusions in general are very poor media for cultivation.

[1] Save on gelatine the cultures were made at 37-38°.

Milk.—When cultivated in milk this streptococcus coagulates it in twenty-four to forty-eight hours.

Agar.—On agar which has been inoculated from blood, for example, one sees after twenty-four hours some small, round, greyish-white, punctiform colonies, which have no tendency to run together later to form a uniform layer. After thirty-six or forty-eight hours the colonies are still round, but larger, though they do not often exceed the size of a pin's head.

Streak cultures on agar are greyish-white, slightly shining, and always very narrow.

Gelatine.—In gelatine at a temperature of 20° there form after twenty-four hours very fine rounded colonies, appearing like so many bluish-white points ; after two, three, or four days the colonies increase slightly in size, and project a little above the surface of the medium, at the same time becoming whitish and opaque. The gelatine is not liquefied.

In stab cultures this streptococcus grows all along the needle track, forming small rounded colonies ; at the surface very little growth takes place.

At a temperature of from 15° to 18° this microbe grows very little or not at all.

Serum.—On solid blood serum one observes after twenty-four hours small, round, translucent, greyish-white colonies, which are a little shining, and have more tendency to run together than those on agar.

Potato.—The streptococcus obtained from the fresh lesions does not grow on potato.

INOCULATIONS.

Mouse.—The mouse, and especially the white variety, is the best subject for experiment. A trace of culture, or of pulp from a hepatised lung, when inoculated under the skin generally kills the animal in forty-eight hours with the following lesions.

At the point of inoculation one observes a marked tumefaction formed by a lardaceous, grey, purulent tissue, surrounded by a reddish, more or less abundant, gelatinous œdema. The lymphatic glands are hypertrophied and indurated. The spleen is hypertrophied, soft, and dark in colour. The liver is ordinarily congested and friable, as are also the kidneys. The blood is black and uncoagulated, the lungs hyperæmic. In the purulent material from the point of inoculation, in the liver, kidneys, blood, and especially the spleen, one can easily stain by Gram's method the microbe in the form of diplococci, often with a clear ring round them. Cultures in peptonised bouillon give superb streptococci.

Guinea-pig.—Subcutaneous inoculation with 1 or 2 cubic centimetres of culture determines a tumefaction and an elevation of temperature, but these phenomena are transient ; the guinea-pig almost always survives.

The same is not the case if the inoculation is made into the peritoneum with streptococci taken from the lungs of pneumonic subjects. One cubic centimetre taken from the bottom of a tube generally suffices to kill a guinea-pig of 400 grammes in from twenty-four to forty-eight hours. At the *post-mortem* examination one remarks some redness of the subcutaneous connective tissue. The peritoneum is

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very much inflamed, and contains several cubic centimetres of an albuminous, turbid, sometimes reddish liquid, in which are floating some flocculi. False purulent membranes of a whitish-yellow colour cover the abdominal viscera. The intestines are usually much congested and filled with gas. The spleen is hypertrophied, soft, and dark in colour ; the liver is friable and full of blood ; the kidneys are soft, and violet in colour. The blood is dark, uncoagulated ; the pericardium contains a clear liquid ; the lungs are congested.

Staining of the blood or pulp of the organs by Gram's method shows the presence of diplococci ; cultures in bouillon give long streptococci.

Rabbit.—Although subcutaneous inoculation in the rabbit determines only a local irritation accompanied by a notable elevation of temperature, this animal is more sensitive than the guinea-pig.

Intravenous inoculation is the best method of proceeding.

If one chooses the marginal vein of the ear, and a certain quantity of culture escapes under the skin, one observes during the following day a little redness and œdema, but in no case is there any erysipelas comparable to that obtained in the same conditions with the streptococcus pyogenes.

The fatal dose for the rabbit is about 1 cubic centimetre from the bottom of a culture of eighteen or twenty hours. The subject of experiment dies in from one to four days.

At the *post-mortem* examination one finds the intestines and also the lungs almost normal. The spleen is but little altered ; one observes only an increased softness of its pulp ; the liver is generally congested and very friable ; the kidneys also appear hyperæmic. The blood is dark in colour and coagulates badly ; the pericardium contains a rather clear liquid.

As in the mouse and guinea-pig, the tissues contain the microbe in the form of diplococci, while cultures invariably give the streptococcus [1] form.

Horse.—The horse reacts well to inoculation with the streptococcus. When introduced under the skin of old horses, in the dose of 2 cubic centimetres, it determines a slight inflammatory swelling, but in young subjects the resulting inflammation is sometimes enormous, especially if the culture is injected under the skin of the chest ; after five or six days an abscess forms, and the pus drawn from it is tenacious, creamy or serous, and rich in diplococci.

The adult horse sometimes supports considerable doses of culture into the veins,—2, 3 up to 400 cubic centimetres ; the temperature rises up to 40·5°; the general state is bad, and the animal emaciates, but it ultimately returns to the normal.

The other animals — pigeon, fowl, dog, pig, sheep, ox—may be considered practically refractory, unless one augments considerably the dose and the virulence of the microbe.

VARIATIONS.

These morphological and biological characters are those which are usually encountered, but some important variations have to be noted.

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[1] The diplococcus form is, I believe, mainly ascribable to the crushing which one is obliged to employ in spreading the tissues on the cover-glasses ; in fact, if one makes sections of the same tissues one often finds the cocci in short chains.

It is very well known that bacteria do not always exhibit absolutely the same cultural characteristics, or identically the same degree of virulence. But the streptococci, and particularly the one with which we are now concerned, are from this point of view very remarkable.

The artificial media of culture have not an invariable composition. Thus one sometimes sees the streptococcus produce a complete turbidity of bouillon, fail to cause an acid reaction, or give short chains ; or it may grow on agar in the form of extremely small colonies, while another may give colonies up to the size of a millet seed or larger.

Sometimes the microbe refuses to grow in gelatine or even on agar, while a good culture may be obtainable in peptonised bouillon.

Nothing is more variable than the pathogenic power of this microbe, and from this point of view the rabbit seems to me particularly interesting.

As I have already said, it generally dies in from one to four days with the lesions I have just indicated, but sometimes death is delayed until the tenth or fifteenth day. The subject then becomes obviously emaciated, its muscles atrophied, the hair erect, and the eyes sunken in the orbits, while there is paresis of the hind quarters, or even paralysis.

In the way of lesions one finds very little. The spleen is normal, the liver is a little congested, the kidneys are normal in appearance, and also the intestine. Frequently there is purulent pericarditis ; one observes also, but rather rarely, inflammation of the joints. In that case even the cultures may remain sterile.

In inoculating direct with blood, pleuritic liquid, or kidney of a horse dead of pneumonia, or with a very fresh culture, one sometimes kills in less than twenty-four hours with feeble doses, from a few drops up to a quarter or half a cubic centimetre.

The lesions are then very different. From the natural orifices (nose and rectum) a sanguinolent liquid escapes. The subcutaneous connective tissue is red ; in the abdominal cavity one may find a large quantity of more or less deeply tinged sanguinolent serosity ; the intestines are red ; the spleen is hypertrophied, soft, and dark in colour ; the liver congested or cooked in appearance ; the kidneys are very friable and red ; the lungs are congested ; the blood more or less fluid, dark in colour, and rarely coagulated ; in the pericardium one encounters an abundant, currant-red liquid, or red blood.[1]

This streptococcus, even without passing it through animals, may have a feeble, medium, or exalted virulence. Moreover, one can in succession give to the same streptococcus these three grades of virulence, either by reducing the pathogenic power of the exalted microbe, or, on the contrary, exalting by successive inoculations the enfeebled microbe.[2]

But it is not only the histo-chemical properties that are variable ; thus, in certain cases the streptococcus shows itself capricious and

[1] Septicæmic lesions are not the expression of particular properties inherent in the microbes, but are the unique form of reaction of the body against a terrible invader. The most different microbes when given a great virulence—a virulence which might be termed the septicæmic virulence—determine in animals lesions that are macroscopically and microscopically identical. Hence, to base a classification on septicæmic characters would be a certain way of leading to error and confusion.

[2] It is easy to obtain the same results with the *streptococcus pyogenes* of man.

stains badly by Gram's method ; in the same preparation one some-times sees chains in which only one half has retained the colour by Grams' method, while the other is intensely stained, red for example.

COMPARISON BETWEEN THE BACTERIUM OF SCHÜTZ AND THE STREPTOCOCCUS PNEUMO-ENTERITIS EQUI OF MM. GALTIER AND VIOLET.

After having obtained these results, it was logical to compare this streptococcus with the microbes already described as specific for pneumonia, and in order the better to carry out this task I requested M. Schütz on the one hand, and M. Galtier on the other, to be good enough to send me the microbes which they had described in their memoirs on infectious pneumonia.

With a good grace, for which I desire to thank them, these two learned professors acceded to my request, and M. Kasparek, of Vienna was also good enough to send me a culture of the bacterium of Schütz.

After reading the memoirs of Schütz, and of Galtier and Violet, with regard to the characters of their microbes, I thought that they could have found only what I myself encountered, and experimenta-tion has entirely confirmed me in that view.

The morphological and biological properties of these microbes are perfectly similar. Contrary to what has been written everywhere in France, the ovoid bacterium of Schütz, like the streptococcus of Galtier and Violet, stains by Grams' method ; moreover, M. Schütz, to whom I remarked this, candidly admitted to me that he had been mistaken on that point, and that the error had been recognised in Germany.[1]

The bacterium of Schütz is a streptococcus of which the histo-chemical properties, the cultures, and the inoculations are exactly those of the streptococci obtained by Galtier and myself.

IDENTITY OF THE BACTERIUM OF SCHÜTZ, AND THE STREPTO-COCCI OBTAINED BY GALTIER AND MYSELF, WITH THE STREP-TOCOCCUS OF STRANGLES.

From the outset of this investigation, when M. Pécus, with a constancy and zeal which I readily acknowledge, furnished me with pathological products and detailed histories of the subjects, I was struck with the resemblance between the streptococcus of pneumonia and the streptococcus of strangles ; and I had even concluded that the outbreak at Saint Cyr was a thoracic manifestation of strangles.

However, I soon saw that the Saint Cyr microbe was to be found everywhere, and a new question then presented itself, namely, that of the identity or duality of the streptococci.

Clinical observation itself soon furnished me with pretty strong proofs in favour of the identity.

All those who have had experience with infectious pneumonia have doubtlessly observed the frequency of sore throats and bronchitis at the beginning and at the end of the outbreaks; that is a point about which there is no dispute. Some experienced practitioners

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[1] From this point of view I cannot too strongly recommend the employment of Gram-Nicolle (carbolised violet), which adds to a great rapidity of execution a precision which is almost mathematical, and which is never obtainable by the ordinary Grams' method.

believe that at its outset infectious pneumonia always presents itself as a benign catarrh of the upper respiratory passages. Be that as it may, I was able to note that in one of the outbreaks manifestations of strangles preceded or accompanied the appearance of the pneumonia.

On the other hand, in an outbreak of pneumonia it is not rare to see some subjects exhibit in the course of the disease, and particularly at the time when the temperature falls, and when there is nothing to indicate strangles, one or several submaxillary abscesses. These abscesses generally indicate an early resolution; the temperature generally commences to fall, and the victorious organism circumscribes the influence of the bacterial agent, which, previously septic, now becomes pyogenic. Now, the pus of these abscesses, which one cannot differentiate from that of strangles, encloses, like the latter, a great number of streptococci which stain by Grams' method. In such a case it is very difficult to admit that a strangles affection has become grafted on a pneumonia, when one observes a decline in the disquieting symptoms.

Moreover, I have been able to produce with the bacterium of Schütz, with the streptococcus of Galtier, and with the one isolated by myself, streptococcus pus absolutely identical with strangles pus. The following is one of these experiments :—

On the 12th June 1896, a horse, ten years of age, received under the skin, with all the necessary aseptic precautions, 1 cc. from the bottom of a tube in which the bacterium of Schütz was growing.

On the 13th there was a flat, hot, sensitive tumefaction, of the size of the hand, and situated under the point of inoculation. Temperature, 37·9°.

On the 13th the tumefaction had disappeared, and there remained only a rather firm, sensitive tumour of the size of a large almond.

On the 16th I withdrew a little pus from this small abscess which had formed at the point of inoculation, and with it I inoculated some peptonised bouillon, which gave me superb streptococci, and some agar, which remained sterile. This pus, when spread on a cover-glass and stained by Grams' method, showed diplococci and some very small chains.

On the 18th June I injected under the skin of the same horse 35 cc. of culture obtained from the first inoculation.

On the 23rd June an abscess as large as the fist had formed at the point of inoculation; the pus which it contained was whitish-yellow, and yielded a rather large proportion of serous liquid. When stained by Grams' method this pus showed diplococci and some masses of cocci, but very few small chains.

On the 26th June I again took some pus from this abscess; it was now quite creamy and very tenacious, and contained almost exclusively long streptococci staining well by Grams' method. I again found streptococci in this pus on the 17th July 1896.

This experiment proves that a transformation of streptococci into diplococci is possible.

One may obtain the contrary proof with a strangles streptococcus whose virulence has been exalted by successive passages through mice. After the fourth inoculation the organs of this animal show by Grams' method only diplococci ; and when the microbe is sufficiently

virulent to kill a guinea-pig of 400 grammes in from fifteen to eighteen hours, with 1 cc. of culture into the peritoneum, subcutaneous inoculation of the horse gives an abscess with diplococci.

If the strangles streptococcus, such as one obtains from abscesses, differs in some respects from the streptococcus found in cases of pneumonia, these differences are due only to a slighter degree of virulence.

Moreover, the strangles streptococcus itself presents very considerable variations in its morphological and biological properties. The streptococcus from some samples of pus grows slowly even in peptonised bouillon; other samples of pus give a very feeble culture, and some even refuse to grow on gelatine; but after a series of generations one obtains colonies on bouillon, agar, gelatine, milk, and serum, which one cannot differentiate from the cultures obtained with the streptococcus of Schütz, or that of Galtier or myself.

I have already said that one can recognise in the streptococci obtained from cases of pneumonia a feeble, medium, or strong degree of virulence. The strangles streptococcus almost always possesses the feeble virulence; I say "almost always" intentionally, for certain grave cases of strangles furnish at the outset a streptococcus of medium virulence.

When the virulence of this streptococcus is diminished, mice will survive four, six, eight, twelve days or more; and the guinea-pig and the rabbit will resist subcutaneous inoculation. It even sometimes happens that 1 cc. of culture will not suffice to kill the former by intraperitoneal, or the latter by intravenous, inoculation. But—and this is very important—after two or three successive passages, or when one is dealing with a strangles streptococcus that has a medium virulence at the outset, one can kill the mouse, rabbit, and guinea-pig in absolutely the same conditions, and with the same lesions, as with a streptococcus of pneumonia.

When inoculated into the subcutaneous connective tissue on the chest of the foal, the bacterium of Schütz, the streptococcus of Galtier and Violet, and my own streptococcus, just like the strangles streptococcus, often produce an enormous inflammatory swelling, followed by a large abscess. In submitting my inoculated subjects to M. Humbert, who has acquired great competence in these matters through his experience with MM. Nocard and Roux, I observed that he did not perceive any difference, no matter what was the source of the streptococci used for inoculation.

SERO-THERAPEUTIC DIAGNOSIS.

Of all the proofs which it is possible to give for or against the duality of bacteria, notably the streptococci, sero-therapeutics is the most decisive.

While the physical and histo-chemical characters and inoculations seem to demonstrate the absolute identity of certain micro-organisms, a study of their toxins and of the curative or preventive sera derived from them may prove their duality.

I may here recall the advantages which I derived from diagnostic sero-therapeutics in identifying the streptococci found in equine purpura hæmorrhagica and the streptococcus pyogenes of man, and in

demonstrating that the latter microbe is distinct from the strepto-coccus of strangles and of pneumonia.

It was thus of interest to determine whether, inversely, a serum obtained with the strangles streptococcus would manifest an influence on this streptococcus, and on those of Schütz, Galtier, and myself, and yet remain without effect on the streptococcus pyogenes.

With this object I used a dog which, during eight months, had received successively increasing doses of strangles streptococci. I had to wait more than six months before I was able to notice a slight but constant influence of the serum of this dog on the streptococcus used to generate the serum. The last bleeding, which was made after eight months of immunisation, furnished me with a serum that was dis-tinctly protective but still only feebly curative, not only—and this is very interesting—against the strangles streptococcus, but also against mine, those of Schütz and Galtier, and others obtained from human scarlatina and diphtheritic false membranes in children.[1] On the other hand, this serum proved to be without effect against the virulent streptococcus of Dr Marmorek.

This dog, unfortunately, died, and I had therefore to suspend my investigations, but, thanks to MM. Nocard and Roux, who have been good enough to place several horses at my disposal, I hope soon to be in a position to complete them.

Furthermore, with regard to Dr Marmorek's serum the streptococci obtained from strangles lesions or from those of pneumonia comport themselves as if they were identical, just as they do with regard to the anti-strangles serum. This observation, I repeat, is of the highest importance, for it constitutes the most tangible proof of the identity of the ovoid bacterium of Schütz with my streptococcus, with that of MM. Galtier and Violet, and with the strangles streptococcus.[2]

In his article on the subject Dr Schütz sought to differentiate his ovoid bacterium from certain microbes that are manifestly distinct from it, such as Friedlander's pneumo-bacillus and Fränkel's pneumo-coccus, but he does not for a single moment compare it with the strangles streptococcus. It is thus very curious to observe the father of the strangles streptococcus failing to recognise his own child in the ovoid bacterium of pneumonia. This is all the more striking when one studies Schütz's two memoirs, for the author employed exactly the same technique, the same cultures, and the same animals for experiment in his researches on strangles and in those on pneumonia.

It thus happens that Dr Schütz's investigations, which ought to have thrown a clear light on the etiology of the pneumonias of the horse, had just the contrary effect, and produced such a degree of confusion that many authors have in good faith described the same microbe under different names, thus involuntarily creating a profound obscurity in our knowledge of the pathology of pneumonia.

At the beginning of this article I intentionally insisted upon the characters attributed to their microbes by Schütz, Delamotte and

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[1] To be quite exact, I ought to add that I have found streptococci that were not influenced by either the one or the other of the two sera here referred to. Are there still other varieties? That is a question which future researches must answer.

[2] Hell, and afterwards Foth, sought to demonstrate the identity of the ovoid bacterium of Schütz with the strangles streptococcus, but their investigations were incomplete. They even committed the error of considering the two species just mentioned as identical with the strepto-coccus of human erysipelas.

Chantemesse, and Galtier and Violet. It will have been noticed how these appear to differ. Those assigned by Schütz are so minute that it was only logical on the part of Delamotte and Chantemesse on the one hand, and of Galtier and Violet on the other, to publish their memoirs. These authors were justly struck with the morphology of their microbe, which they termed a streptococcus—that is to say, the only name that could rightly be applied to it.

The first of these authors at the very outset fell in with a microbe of great virulence, hence the rather special characters which they assigned to it. The second often obtained their streptococcus from food-stuffs, hence the facility with which they were able to cultivate it on vegetable media, notably potato. It ought also to be observed that at that period the authors could not absolutely establish the identity of their streptococci, and this certainly led them to study various species. But, I repeat, in view of Schütz's article their investigations were perfectly logical.

The streptococcus of strangles is a very widely disseminated organism. It is present in almost all discharges, and food materials of both good and bad quality frequently harbour it, as does also the intestinal tube, litter, etc. I believe that with regard to this point Galtier and Violet have not been guilty of any exaggeration.

But the biological properties, and notably the virulence, of this streptococcus vary greatly according to the source from which it is drawn. As it is obtained from the intestine it is often possessed of little or no pathogenic power; the same is the case with the streptococcus found in food, and sometimes in that present in certain discharges, and it is then necessary to have recourse to some devices of inoculation to render it virulent.

The strangles streptococcus persists for months in the natural cavities, and especially in the nasal cavities of the horse. It may even, as I have observed in the case of an experimental horse, exist in the body for a long time without appearing to inconvenience its host. For example, a mare which received an intravenous injection of streptococci was at first ill and then appeared to make a complete recovery, but a month afterwards cultures from the kidney of this animal yielded strangles streptococci.

In practice one distinguishes a great variety of pneumonias. Schütz, on the ground of his researches in 1887 established the unity of equine pneumonia. I have made observations in the same sense, and I have been able to observe the presence of the same streptococcus in animals attacked with diseases of the chest to which my colleagues applied such different names as strangles pneumonia, pneumonia due to cold (*a frigore*), contagious pneumonia, stable pneumonia, pleuro-pneumonia, pleurisy, typhoid pneumonia, pleuro-enteritis, broncho-pneumonia, influenza, etc.

Among the public, and even among veterinary surgeons, the view that a horse suffers only once from strangles is very widely spread. There appears, however, to be a contradiction between this opinion and the observation that the streptococcus of Schütz is present in the viscera and even in the blood of pneumonic patients. But, as will presently be seen, this contradiction is only apparent. It is true that the horse which has recovered from strangles has acquired a certain resistance towards the streptococcus of Schütz, the proof

being that if at the same time one inoculates this animal and another of the same age that has never had the disease, with a culture of the microbe in question, only a slight and very transient tumefaction forms at the point of inoculation in the former, while in the latter one observes on the following day the presence of a large inflammatory swelling, followed later by an abscess. In the same way, when one repeats injections of the strangles streptococcus into the subcutaneous tissue of a horse the dose and the virulence of the microbe must be increased each time if one wishes to obtain an abscess.

But in proportion to the time that has elapsed since the animal has recovered either from an inoculation or from a natural attack of strangles the body loses its powers of resistance, until new inoculations are followed by effects more considerable than the first. Moreover, the immunity conferred by a first attack is quite relative, and not at all comparable to that of vaccination, for example, for animals quite recently recovered from an attack of strangles support badly inoculations with very virulent strangles streptococci.

The experience gained in practice is not in disagreement with these facts. We pretty frequently see horses attacked several times with strangles, and very old subjects sometimes show us the same clinical signs of strangles as young horses, although it is very probable that those animals have had strangles in their youth.

Frequently also young horses contract a pueumonia when they appear to have recovered from strangles, but when they in reality still harbour numerous streptococci, as my bacteriological researches have demonstrated.

Thus, the streptococcus of Schütz confers only a relative immunity as the result of a first attack, and this immunity is inversely proportional to the time which has elapsed since the disease or the inoculation.

M. Pecu has been good enough to furnish me with the previous history of his pneumonic patients. The following is the very interesting table which he has sent me:—

No.	Age.		
1	11 Years.	Submaxillary strangles abscess . .	at 4 Years.
2	13 ,,	Strangles	,, 4 ,,
3	13 ,,	Strangles sore throat	,, 4 ,,
4	16 ,,	Strangles	,, 4 ,,
5	6 ,,	Do.	,, 4 ,,
6	11 ,,	Strangles sore throat	,, 4 ,,
7	7 ,,	Do. do . . .	,, 3 ,,
8	11 ,,	Strangles	,, 4 ,,
9	12 ,,	Do.	,, 6 ,,
10	11 ,,	Strangles nasal discharge . . .	,, 4 ,,
11	8 ,,	Strangles	,, 3 ,,
12	9 ,,	Do.	,, 6 ,,
13	7 ,,	Do.	,, 4 ,,
14	8 ,,	Do.	,, 3 ,,
15	9 ,,	Sore throat and strangles pneumonia	,, 6 ,,
16	13 ,,	Strangles	,, 4 ,,
17	16 ,,	Do.	,, 3 ,,

I ought to observe that in the foregoing part of this article I have not been concerned at all with the etiological rôle of the streptococcus of strangles, which is dealt with in the following part.

CONCLUSIONS.

1. The pneumonia bacterium of Schütz is a streptococcus, and it is no other than the streptococcus of strangles.

2. The streptococcus of Delamotte and Chantemesse, the streptococcus pneumo-enteritis equi of Galtier and Violet, and with great certainty the cocci or streptococci described before or after Schütz's work, are identical with the bacterium of the last-named author, that is to say, with the strangles streptococcus.

3. All these stain by Grams' method.

4. The strangles streptococcus is biologically different from the streptococcus of human erysipelas, or rather, we ought in the meantime to separate the streptococci into two great groups which may be encountered either in man or animals, the one corresponding to the streptococcus of erysipelas, and the other to the streptococcus of strangles.

5. The streptococcus of Schütz (read bacterium of Schütz or the strangles streptococcus) is found in the organs or pathological products of horses attacked with the so-called chill pneumonia, infectious pneumonia, stable pneumonia, strangles pneumonia, pneumo-enteritis, pleuro-pneumonia, pleurisy, broncho-pneumonia, and influenza.

It frequently exists in the intestinal contents, forage, litter, and dung.

6. A first inoculation with the strangles streptococcus seems to confer a certain resistance against a later inoculation, but this does not amount to a solid immunity, and solipeds in particular always remain susceptible to this streptococcus.

7. The true pathological rôle of the streptococcus of Schütz has yet to be determined.

PART II.

Hitherto the symptoms which have been considered characteristic of influenza of the horse are the following—intense fever, torpor, staggering gait, eyes half closed and weeping, mahogany colour of the visible mucous membranes, and contagion.

These are, in effect, the classical signs of this affection, and if they were constant the diagnosis of it would not offer any difficulty. Unhappily, that is not the case. Although, when the disease attacks a large stud of horses one almost always finds some subjects presenting the pathognomonic symptoms, one or other of these symptoms is often absent in a large number of the animals, which therefore present an entirely different clinical feature. If we join to the symptomatic varieties the multiple names given to this disease, such as typhoid fever, influenza, epizootic gastro-enteritis, etc., with the various possible complications, one can easily understand that veterinary surgeons are not very skilful in diagnosing influenza.

As a matter of fact, one may say that the actual state of our knowledge on this affection forms such an imbroglio that some have even

gone so far as to deny the existence of the disease as a distinct morbid entity.

Only a knowledge of the specific agent of the disease can clear up the situation, and gradually determine the domain of equine influenza.

Last year M. Nocard, knowing that I was actively occupied with this question, sent me to study a grave contagious disease of a typhoid character which prevailed in Paris among the horses at the slaughter-house.[1]

From the specimens which were sent to me, and from those which I collected, I isolated a special microbe, of which I have studied the morphological and biological characters.

Some months later M. Blanchard was good enough to send me some fresh specimens from horses that had died very suddenly from influenza or infectious pneumonia. I did not omit to search for the micro-organism found in the previous outbreak, and I had the satisfaction of discovering it in almost every case.

At this stage, when my researches threatened to prove sterile owing to the want of horses for experiment, I learned that four horses of the riding school at the Alfort Veterinary College were about to be cast. These animals were about to be sold to the butcher. · The occasion appeared to me to be an excellent one for experimenting on horses, and the Director of Agriculture, to whom I explained my investigations, did not raise any objection to my receiving the four cast subjects. To this enlightened assistance I owe in great measure the success of my studies on infectious pneumonia.

In effect, the horses inoculated with the microbe obtained from the subjects of influenza showed the most characteristic symptoms of the classical disease. I therefore considered this microbe as the specific agent of influenza of the horse, and since then I have been able to demonstrate its presence, not in every case (I shall explain why immediately), but at least in some subjects in every outbreak.

MORPHOLOGICAL AND BIOLOGICAL PROPERTIES OF THE MICROBE OF INFLUENZA OF THE HORSE.

It is a cocco-bacillus, scarcely so large as the germ of fowl cholera, and belonging, with the latter and the bacteria of buffalo disease, septicæmia of rabbits and ferrets, swine-plague, etc., to the genus Pasteurella of Trevisan (1887). It is non-motile, and it stains pretty well with gentian-violet and fuchsin, but not at all by Gram's method.

Hydro-alcoholic or carbolised fuchsin has the inconvenience of frequently leaving a precipitate and dirtying the preparations. The following solution of fuchsin has not this inconvenience, and, moreover, it has the advantage of staining excellently in four to six seconds. Mix together and agitate: distilled water, 10 parts; essential oil (a final product in the distillation of alcohol), 1 part; retain only the aqueous portion, and add to it a fifth of its volume of alcohol at 95°. After that it remains only to dissolve the fuchsin

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[1] In this outbreak the mortality was considerable. Out of twenty-one attacked ten died—three from pleuro-pneumonia, and the others with cardiac and intestinal lesions, chiefly the latter.

in this alcoholic solution in order to obtain the required stain. The fuchsin ought to be added in such a proportion that part of it will remain undissolved at the bottom of the vessel—that is to say, the solution ought to be saturated. It does not alter when kept in a stoppered bottle. Before putting this stain on the preparation, it is advisable to throw on the latter some drops of a mixture of alcohol and ether in equal parts.

When one examines the preparation after it has been stained, the bacteria present themselves under the form of very small diplococci, isolated cocci, and longer forms with rounded ends. These last cocco-bacilli represent the true form of the microbe. At the moment of division they appear as diplococci, while the micrococci result from the complete and recent separation of diplococci.

The microbe also often appears as a diplococcus when it is examined without staining.

In some special conditions the cocco-bacillus elongates and presents itself under the form of a genuine small bacillus. Finally, in the pus of experimental abscesses obtained by subcutaneous injection in the horse, it takes the form of streptococci-bacilli.

It does not form spores, and it is killed in less than a quarter of an hour at 65°.

CULTURES.

It is an ærobic organism. Its first cultures are difficult to obtain, but when it has been accustomed to artificial media it grows rather abundantly.

In peptonised bouillon, after twenty-four hours, one observes a uniform turbidity, which lasts for several days. The reaction of the medium does not change.

Plain bouillon is not so favourable for growth.

On gelatine, at a temperature of 20°, it forms after two or three days rounded colonies which are at first transparent, and then slightly opaque and white, and which never liquefy the medium. They scarcely exceed a millet seed in size, and resemble rather closely the colonies of zoogleic tuberculosis, save that the latter are much less firmly adherent to the gelatine.

Agar is a rather poor medium of culture, whether it be plain or glycerinised. If the material used to inoculate is vigorous or abundant, transparent iridescent colonies appear on the surface of the agar, and gradually become opaque and bluish white.

The germ of influenza grows in milk, which is not coagulated even after several weeks.

Agar of Würtz and rubin agar are not reddened.

Potato is not suitable for the cultivation of this microbe.

Of all media the most favourable is peptonised bouillon to which a small quantity of serum has been added.

SEARCH FOR THE MICROBE.

Inoculation and the direct cultivation of pathological products taken from horses affected with the disease as a rule do not give any result in ordinary conditions, but if one takes 4 or 5 cc. of blood, pleuritic liquid, or pulmonary serosity, and injects the material into

the peritoneum of a guinea-pig, the animal not rarely succumbs to a peritonitis, in the liquid of which the microbe of influenza is encountered in considerable numbers. Nasal discharge, whether sanious or not, taken especially at the beginning, still more frequently gives a positive result. If the culture is not pure one inoculates in succession, into the peritoneum, one, two, or three guinea-pigs, and very soon only the bacillus of influenza shows itself.

This microbe retains its virulence for a long time in peritoneal liquid, and when transferred thence to artificial media it grows in these abundantly.

EXPERIMENTAL INOCULATIONS.

When one wishes to make experimental inoculations much the best plan is to use the rich cultures obtained from the peritoneum of the guinea-pig. This culture is very virulent, since $\frac{1}{2}$ cc. inoculated under the skin of the horse is capable of killing it in twenty-four to forty-eight hours.

The guinea-pig, rabbit, and mouse are relatively very sensitive to the bacillus of influenza.

Guinea-pig.—When injected under the skin 1 cc. of bouillon culture or a few drops of culture from the peritoneum will kill a guinea-pig in twenty-four to forty-eight hours.

A large, hot, firm, sensitive swelling forms at the point of inoculation ; the hairs of the animal become erect, it is very depressed, and the eyes are dull. The temperature very quickly rises to 40°, 41°, or more.

At the autopsy the connective tissue is reddened, and at the point of injection one finds a purple swelling surrounded by a reddish inflammatory œdema. The centre of the tumour allows the escape of a material which is slightly greyish and sanguinolent, sometimes purulent. The spleen is slightly hypertrophied and soft ; the liver and kidneys are congested ; the pericardium often contains a rather abundant, clear and colourless liquid ; the lungs are normal or hyperæmic ; and the blood is dark and coagulates badly.

If the inoculation is made into the peritoneum, with a few drops of bouillon culture or a trace of culture from the peritoneum, death supervenes in eighteen or twenty-four hours. When the dose is increased the subject dies in six or eight hours. By way of macroscopic lesions one finds an intense peritonitis with a large quantity of liquid which is slightly milky, very thin, coagulable, not at all mucoid, and remarkably rich in the cocco-bacilli of influenza. In the peritoneal liquid one observes very little purulent false membrane, and generally none on the viscera.

The other lesions are those of a violent septicæmia—connective tissue red, intestines, liver, and kidneys congested and friable. The spleen is notably congested and livid in colour.[1]

Rabbit.—The same doses as those indicated above for the guinea-pig are applicable to the rabbit, which appears to me to be even more sensitive than the former animal to this bacillus.

[1] Successive passages in the guinea-pig considerably augment the virulence of the cocco-bacillus for this rodent, but diminish it for the horse. Thus, after a hundred passages in the guinea-pig an influenza bacillus which killed the horse in twenty-four hours by subcutaneous inoculation no longer causes death even when one injects 4 cc. into the veins.

By subcutaneous injection this microbe determines a very painful swelling, and the animal soon becomes unable to support itself on the inoculated leg. It retreats into a corner and dies in twenty-four or forty-eight hours without having taken any food. The temperature rises to 41° or even 42°.

The autopsy shows lesions identical with those of the guinea-pig, but more frequently than in the latter one finds peritonitis or pleurisy with abundant false membranes.

When the inoculation is made into the muscles of the thigh the muscular fibres undergo the vitreous degeneration, and here and there one finds black tumours.

Whether it be the case of a rabbit or a guinea-pig, the lymphatic glands adjacent to the point of inoculation are hypertrophied, œdematous, and hæmorrhagic.

Mouse.—The mouse always dies in twenty-four or forty-eight hours after subcutaneous or intraperitoneal inoculation. After the injection of some drops of bouillon culture into the subcutaneous connective tissue the animal rapidly becomes somnolent, the respiration very hurried, the eyes closed and weeping, the hairs erect. At the point of inoculation there forms a hot sensitive swelling, resembling rather closely, even at the autopsy, that determined by the streptococcus of Schütz.

At the *post-mortem* examination one finds the spleen hypertrophied and soft, the liver congested, and the blood dark in colour. Intraperitoneal inoculation with a trace of culture from the peritoneum kills the mouse in eight or ten hours.

Dog and Cat.—A quarter of a cubic centimetre of peritoneal liquid from a guinea-pig when injected under the skin of one of these carnivora determines an enormous tumefaction which is very sensitive, and which immobilises the whole limb. The appetite is lost and the temperature rises.

The cat is much the more sensitive of the two. Intravenous inoculation with a dose of ½ to 1 cc. kills the animal with hæmorrhagic lesions, which are sometimes very remarkable. The blood and the muscles are almost black.

Sheep.—When inoculated in the same conditions as the dog the sheep more frequently succumbs to subcutaneous inoculation, and always when the inoculation is intravenous. The temperature reaches or exceeds 41°. Immediately after intravenous injection the general symptoms are very alarming.

The autopsy shows, after intravenous inoculation, petechiæ on the diaphragm, mesentery, lungs, and heart ; the spleen is a little soft, the liver has a markedly cooked appearance, the kidneys are purple and soft. The intestines appear normal. In the thoracic cavity one finds about a quarter of a litre of citron coloured liquid. Neither the blood nor the muscles are abnormally dark in colour.

If the inoculation has been made under the skin one finds at the point of injection an enormous inflammatory swelling.

Young Pig.—Subcutaneous inoculation of ¼ cc. of culture from the peritoneum determines a reddish œdema, and a rapid elevation of temperature, which rises to 40·5°. One may also observe some red spots on the body, but after the second or third day recovery is complete.

Ox.—Subcutaneous inoculation of ½ cc. of culture from the perito-
neum did not determine either local or general reaction.   However,
the animal died fourteen days afterwards in my absence.

Ass.—Subcutaneous inoculation determines a very intense local
reaction ; on the other hand, the general state, and the temperature
especially are, as in the ox, little modified, but notwithstanding this
the subject sometimes dies on the fifth day with lesions of pleurisy
and peritonitis.

Horse.—The horse is very sensitive to this microbe.[1]   Intravenous
inoculations of 1 to 2 cc. of culture from the peritoneum may entail
death in some hours. A short time after injection the subject presents
grave general symptoms, the pulse is quick, small, thready, and im-
perceptible, while the beats of the heart are strong and quick ; the
respirations are accelerated to 35 or 40, and the temperature quickly
rises to 41°, 41·5° or more.

One also observes muscular tremors which become general, the
appetite is entirely lost, and the animal is very dull. The mucous
membranes assume a mahogany hue, the eyes appear swollen and
are sometimes weeping, and frequently the patient exhibits signs
of enteritis with more or less violent colic.   Pretty frequently
œdema appears at various parts of the body, particularly the legs.

If the animal is going to recover the general symptoms gradually
disappear, but it is not rare to see jaundice or painful synovitis set in
suddenly.   On the other hand, when the patient is about to die the
pulse becomes absolutely imperceptible, sweats cover the body, and
death occurs either during a period of relative calm, or more frequently
during agitation.

At the autopsy the blood is black and uncoagulated, and the
muscles have a cooked appearance ; those of the heart especially have
this tint, and are excessively friable. The liver is purple and much
softened, and the intestinal mucous membrane is congested. All the
viscera are more or less hyperæmic and very friable, with the excep-
tion of the spleen, which is little altered. In the pericardium one
frequently finds some citron-coloured clear, reddish, or turbid liquid.

Some petechiæ are often observed on the viscera, and they are also
found on the serous membranes, which sometimes show traces of
inflammation ; it is thus not rare to observe a commencing peritonitis
or pleurisy with effusion of liquid into the cavities.

Intra-tracheal inoculation determines the same symptons, but it is
much less dangerous than the preceding. The animal resists the
injection of 2 cc. of culture from the peritoneum, although it will
almost certainly succumb if the injection is made into the veins.

After subcutaneous injection of the cocco-bacillus of influenza,
an enormous hot and very sensitive œdema forms at the point of
injection.   If the animal does not succumb an abscess forms, with
sanguinolent liquid and matter containing cellular, tendinous, or even
muscular tissue.

When ½ cc. or ¼ cc. of culture from the peritoneum is injected under
the skin it determines, besides the œdema, stupefaction, unsteadiness,
and difficulty in walking, muscular tremors, marked acceleration of
the pulse, which becomes almost imperceptible, and an elevation of
temperature to 40°, 41°, or 41·5°.   The muçous membranes are

[1] The individual susceptibility is very variable.

mahogany-coloured, and the eyes are sometimes swollen and weeping. If the animal succumbs one frequently finds a degeneration of the cardiac muscle. Young horses are more susceptible than old ones.

White Rat.—The white rat is an excellent subject for experiment. Subcutaneous injection of ¼ cc. of a culture from the peritoneum does not kill it.

Pigeon and Fowl.—These birds are very resistant. They do not succumb except to a large intravenous injection—1 cc. of culture from the peritoneum. The fowl may even withstand this inoculation, but it is always very ill, and its temperature rises to 42·5°.

The search for the microbe in the blood and tissues of the subjects of infectious pneumonia is difficult. In order to succeed, it is necessary to operate with products rich in microbes, to spread them in a thin layer on a cover-glass, without crushing them, and to stain very rapidly (four to six seconds) with fuchsin in the way already indicated. In the tissues the cocco-bacillary form or even the bacillary form is more frequent than in cultures. In horses dead of the disease the tissues are generally so poor in the specific bacteria that it is not easy to see these after staining.

APPARENT INCONSTANCE OF THE COCCO-BACILLUS IN THE TISSUES AND PATHOLOGICAL PRODUCTS OF THE SUBJECTS OF INFLUENZA.

I have just said that intraperitoneal inoculation of suspected products to the guinea-pig constitutes the best method of demonstrating the presence of the microbe, but even this method is frequently unsuccessful. These failures appear to me to have two principal causes:—(1) The small quantity of virus capable of determining serious effects in the animals attacked, and the rapidity with which the microbe disappears or becomes attenuated in the body after it has exerted its toxic action; (2) its association with foreign microbes.

It is especially by means of its extremely formidable toxins that the cocco-bacillus of influenza acts, especially in the horse; but the organic reaction rather easily overcomes the microbe or its virulence, so that if the patient does not succumb rapidly it is rare to encounter, or, to speak more exactly, to be able to demonstrate, the presence of the specific agent. Nothing is more certainly established than that fact. If, for example, one finds the cocco-bacillus in the nasal discharge at the outset of the disease, it is often absolutely impossible to isolate it from the lung, blood, or organs when the animal dies after eight, ten, or fifteen days.

Moreover, some of the horses experimentally inoculated into the muscles with all the necessary aseptic precautions have died as early as the third day, after having manifested the classical symptoms of infectious pneumonia, and yet it has not been possible for me to isolate the cocco-bacillus used to infect them either from the blood, viscera, or pathological products.

On the other hand, the bacillus of infectious pneumonia places the animal organism in an extraordinary state of susceptibility with

regard to extraneous bacteria, notably streptococci. This explains why, having injected aseptically a pure culture of cocco-bacilli, inoculation of artificial media with blood or pulp of the fresh carcase gives exclusively strangles streptococci.

At a later part of the article I shall show that the streptococcus finds in the body of an animal influenced by the cocco-bacillus of influenza an extremely favourable soil for its development, and that after it has become installed there it interferes considerably with the search for the specific microbe.

DIFFERENTIAL DIAGNOSIS BETWEEN THE COCCO-BACILLUS OF INFLUENZA AND SCHÜTZ'S BACTERIUM OF INFECTIOUS PNEUMONIA.

Cocco-bacillus of Influenza.	*Schütz's Bacterium.*
Presents itself under the form of cocco-bacilli smaller than the bacteria of Schütz.	Presents itself as monococci, diplococci, or streptococci, but never as bacilli.
Does not stain by Gram's method.	Stains very well by Gram's method.
Causes general turbidity of the bouillon in which it is growing.	Generally deposits flocculi at the bottom of the tube, while the liquid remains perfectly limpid.
Ærobic.	Æro-anærobic.
Never causes acidity of peptonised bouillon ; does not coagulate milk ; does not cause fermentation of lactose.	Often causes acidity of peptonised bouillon ; coagulates milk ; causes fermentation of lactose.
Grows well in gelatine between 18-20°.	Grows badly in gelatine between 18-20°.
Readily kills the guinea-pig, rabbit, and horse by subcutaneous inoculation.	Does not kill the guinea-pig, rabbit, or horse by subcutaneous inoculation in ordinary conditions.
Is easily transformed into a vaccin.	Is not transformable into a vaccin.
Does not grow in filtered cultures of strangles or pyogenic streptococci.	Grows in filtered cultures of the influenza bacillus.

PATHOGENESIS.

Infection takes place by the ingestion of products soiled with the influenza microbes. Water, which is so easily polluted by the discharge from affected animals, excrement, and other causes, certainly plays a very important rôle.

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On the 15th February 1897 I obtained some of the drinking water from a stud in which influenza prevailed at the time. My investigations enabled me to isolate from this water a very considerable number of species of bacteria, including the cocco-bacillus of influenza.

It does not suffice, however, to introduce cultures of the specific microbes with the food or drink in order to determine the development of influenza ; in this disease, as in a great number of others, there are secondary or predisposing causes, which are not known to us, and which create the epidemic state, under what one may term the latent form.

Moreover, all animals have not the same susceptibility ; for example, among eight guinea-pigs which I fed on the 26th and 27th January and 2nd February with cultures of the bacillus of influenza, three succumbed on the 19th February, while the others were not affected.

The microbe with which these animals were fed could be isolated from only one of them, and in that case it was obtained from the pericardial liquid, where it existed in abundance. This fact is interesting, for the cocco-bacillus employed was obtained from a case of influenza in which the horse died in consequence of pericarditis.

The successes are more frequent when one causes the experimental animal to ingest, along with the bacillus of influenza, some other microbe, such as the streptococcus of strangles.

In the horse I have experimented only on two very old animals ; one of the two, after having consumed a culture of the influenza cocco-bacillus associated with another microbe, at the end of forty-eight hours showed the main symptoms of influenza.

Contrary to what is stated in some of the classical articles, influenza is not always due to contagion, more or less direct, from a diseased animal to a healthy one. I am convinced that the influenza cocco-bacillus can multiply in forage, dung, the soil, water, etc. ; that it may vegetate in these as a saprophyte, and become pathogenic under the influence of causes which entirely escape us.

Influenza does not solely prevail in the epizootic state ; one finds sporadic cases where it is very difficult, if not impossible, to suppose that contagion has been in operation.

I recollect, among other facts, that a two-year-old colt died from undoubted influenza on the farm where it had been bred, although the farmer did not recollect ever having seen such a case, and although the disease was unknown in the district.

None of the so-called sporadic or spontaneous cases of influenza can be explained except on the view that the saprophytic microbe becomes transformed into a pathogenic one.

The point on which I especially desire to insist is that influenza of the horse, like that of man, may attack a single animal in a series ; in other words, in order to have influenza it is not necessary to have an epizootic.

EXPERIMENTS IN VACCINATION AND SERUM THERAPEUTICS.

None of the microbes previously described correspond with the cocco-bacillus which I have just indicated. However, M. Pasteur

made known, à *propos* of the attenuation of viruses, and without attaching any specific importance to it, a micro-organism which he had obtained from the discharge of an influenza horse. Although the biological characters of this microbe were from the present day standpoint insufficiently indicated, they correspond pretty well with those of my cocco-bacillus.

A point in which these two microbes especially approach one another is the facility with which they are transformed into vaccins.

The influenza bacillus when properly attenuated and inoculated into a healthy animal does not provoke any serious accident, but it confers immunity, since an inoculation made a few days afterwards with an otherwise certainly fatal dose does not kill the animal. I have already commenced some protective inoculations on the horse, and although I am only as yet at the experimental period, the results obtained are encouraging.

I am already able to assert that it is possible to obtain a serum which is not only protective, but also curative. A horse which was placed at my disposal by MM. Nocard and Roux has already given me a serum which enables one to save a rabbit that has already received an otherwise certainly fatal dose.

When my researches are more advanced I shall return to the subject of vaccination and serum therapeutics, the practical importance of which is obvious to everybody. And, with a knowledge of the specific agent, the pathology of influenza of the horse will assume a new aspect, and one, I hope, that will be attended with good results.

Lastly, it gives the final blow to the pretended identity of influenza of man and that of the horse.

PART III.

I am aware that my first note on this subject was not received without some misgivings.

Some have remained quite incredulous; others, imbued with the specific nature of the bacterium of Schütz, have seen in my article a claim for the identity of pneumonia and strangles, and have refused to admit that.

These latter would have had some reason for their attitude if in any part of my communication I had said, or even allowed it to be thought, that the bacterium of Schütz was the causal agent of infectious pneumonia.

Assuredly the microbe of Schütz plays only a rôle of association— one which cannot be neglected, but one which is always secondary.

As soon as I recognised the identity of the bacterium of Schütz with the streptococcus of strangles, I acquired the conviction of its etiological insufficiency.

From the purely clinical point of view it was impossible to identify strangles with pneumonia, this last readily prevailing among subjects completely recovered from strangles, although certain veterinary surgeons would say recovery leaves a real resistance against a second pulmonary infection. I know quite well that one sees horses have two attacks or more of pneumonia in succession, but that is excep-

tional, and it must be admitted that very generally a first attack confers immunity.

On the other hand, I have several times observed the absence of the bacterium of Schütz from the completely hepatised lungs of horses which during an epizootic presented the most typical symptoms of infectious pneumonia, and this fact constitutes a very strong presumption in favour of the existence of an unknown causal agent different from the bacterium of Schütz.[1]

Without any preconceived idea, I then sought to discover in the pathological products of the subjects of pneumonia the true specific microbe.

In that I had to overcome numerous difficulties, on which I need not here insist ; suffice it to say, I was ultimately able to demonstrate the presence of a microbe which I soon recognised to be that of influenza.

I have encountered this microbe in cases of pleurisy, pleuro-pneumonia, so-called infectious pneumonia, pneumonia through chill, typhoid pneumonia, strangles pneumonia, stable pneumonia, broncho-pneumonia, and even in cases of infectious sore throat.

This explains why in outbreaks of infectious pneumonia one so frequently observes that some of the subjects present symptoms of pure influenza, perhaps ephemeral, but quite clear.

EXPERIMENTAL RESEARCHES.

It was very important to endeavour to reproduce experimentally the typical lesions observed in outbreaks of infectious pneumonia.

I have abstained from injecting cultures into the lung or thoracic cavity, for even in cases of success the demonstration would have been far from perfect, since lesions of pneumonia, pleurisy, or pleuro-pneumonia may be produced by the intrathoracic or intrapulmonary injection of a host of different species of microbes.

On the other hand, it is perfectly evident that the infection of the lungs and pleura is produced in special circumstances, since natural infection sometimes determines a general disease, and sometimes a localised one, notably in the lungs or the pleura.

Perhaps we may yet succeed in determining exactly all the conditions necessary to produce with certainty such a complication ; meanwhile I desire to make known the result of my attempts to produce pulmonary and pleural infection.

At the outset I may remark that in order to produce such localisation it appeared preferable to employ enfeebled microbes. It has been by using a culture only slightly virulent that I have succeeded in setting up pleuro-pneumonia in the rabbit by inoculating into the muscles of the thigh. The hepatisation of the lungs was such that the tissue had the consistence of liver. I had the opportunity to show this rabbit to M. Nocard and several other veterinary surgeons. Although this experiment has in my opinion a real significance, I do not insist on it, since it was carried out only on a rodent.

The facts relating to the transmission of the disease to the horse will certainly appear much more important. Up to the present time

[1] From the lungs of other horses in the same stable I have obtained with the greatest ease the bacterium of Schütz.

I have mainly employed subcutaneous inoculations in solipeds, and although I have not yet set up pneumonia I have at least obtained quite characteristic pleurisies.

I hope, however, soon to succeed in experimentally setting up pulmonary hepatisation. If I have not yet succeeded, that is, without doubt, due to the fact that the number of my inoculations is still restricted and the age of the subjects has been a little too advanced.

On the 5th June 1897, an aged light draught horse received under the skin ¼ cc. of a culture of the influenza bacillus from the peritoneum.

On the following day the animal presented an extensive œdema at the point of inoculation ; it was dull, the temperature was 39·7°, and the mucous membranes appeared almost normal. Up till the 8th there was nothing particular to note, but on that evening the animal presented some symptoms of pleurisy. It died on the 9th June at 2 P.M., and the *post-mortem* examination was performed immediately.

The intestines were congested in some places, and the peritoneum was manifestly inflamed. The other abdominal viscera appeared healthy or slightly hyperæmic.

The thoracic cavity contained five or six litres of turbid liquid, and the pleura was strongly inflamed ; there was an abundant deposit of false membranes, exactly like an omelette, on it. The lungs showed nothing abnormal, and the heart presented numerous petechiæ.

So much for the clinical examination; which was not without interest ; but the chief interest of this experiment relates to the bacteriological examination. I had injected under the skin of this horse, with the most minute precautions, a pure culture of the influenza bacillus; but in the turbid liquid of the pleura I found, along with a considerable number of the microbes inoculated, also the strangles streptococci. This last observation has the highest importance, for in cases of natural pleurisy or pleuro-pneumonia one almost constantly finds the streptococcus of Schütz. It proves once more with what facility it multiplies within the bodies of influenza patients.

In an old donkey, inoculated in the same conditions, similar lesions were produced, associated with strangles streptococci ; in this case, however, there was less elevation of temperature.

ETIOLOGY.

When one recollects, on the one hand, that the influenza bacillus generally disappears very rapidly from the bodies of patients, or cannot be demonstrated in them ; and that, on the other hand, streptococci, and particularly the streptococcus strangles, multiplies for months on the mucous membranes and in the living tissues, one has the key to the etiology of infectious pneumonia.

It seems to me to result clearly from the investigations just described, that the so-called infectious pneumonia is a manifestation of influenza, in which, thanks especially to the remarkable association of strangles streptococci, a localisation of the disease in the lungs is produced.

At the moment when the animal is influenced by the bacillus of influenza, the streptococcus, which is so widely disseminated, often

already exists in the respiratory passages, without, however, always manifesting its presence by any external signs. Favoured by the organic depression determined by the cocco-bacillus, the streptococcus multiplies in the lungs, forms caseous foci around the bronchi, and then invades gradually the parenchyma, and more or less completely the entire body.

It is curious to observe how easily the specific microbe gives place to the streptococcus, so that soon one can only find the latter. Moreover, as I have demonstrated, even when the influenza bacillus and the streptococcus exist at the same time, very generally the latter only can be put in evidence.

The following are some experiments which prove that conclusively :—

(1) *Inoculations.*—In a certain number of cases where the influenza bacillus is inoculated into the peritoneum of the guinea-pig along with the strangles streptococcus, the latter only is found in the peritoneal serosity. This happens when the streptococci inoculated are very abundant, or the influenza bacillus slightly virulent.

(2) *Cultures.*—If one inoculates a tube of peptonised bouillon at the same time with the bacillus of influenza and about an equal quantity of the streptococcus of strangles or the streptococcus pyogenes, after twenty or twenty-four hours the culture will show only abundant streptococci.

If one inoculates with the bacillus of influenza a culture of the strangles streptococcus or of the streptococcus pyogenes, of four or five days old, which has been filtered through a Chamberland bougie, the liquid remains clear and the influenza bacillus does not grow. On the other hand, the streptococcus pyogenes and the strangles streptococcus in the same conditions grow in a filtered culture of the influenza bacillus. When one uses pulp from the hepatised lung of the horse for inoculating culture media it is mainly streptococci that one introduces, and this explains the error hitherto committed.

Contamination is effected mainly by nasal discharge ; the sick animals spread this in the troughs while drinking, and into the manger by coughing.

Strangles plays a predisposing rôle of the first importance. I have already mentioned that I found the influenza bacillus not only in the bodies of animals suffering or dead from infectious pneumonia, but also in cases of pleurisy, pleuro-pneumonia, so-called strangles pneumonia, typhoid pneumonia, stable pneumonia, and pneumonia from a chill. I return to this point, for it is evident proof of the insufficiency of clinical methods in the diagnosis of diseases of the chest. It is impossible, and certainly inexact, to distinguish between the differently named pneumonias—infectious, strangles, etc.

The pneumonia of influenza is not necessarily infectious ; we see sporadic cases both of pneumonia and of influenza. In a word, the isolated appearance of pneumonia does not exclude the possibility of its being due to influenza, at least that is the outcome of my bacteriological researches.

My researches also prove that if the germ of influenza is not the only microbe capable of causing pneumonia in the horse (as is, indeed, probable) it is at least one of the most frequent causes.

Evidently all influenza pneumonias do not display the same clinical

picture, there being many causes to modify the apparent symptoms, and one easily understands how the disease has been subdivided to classify its variations.

Be that as it may, the term influenza pneumonia is the only one suitable for the so-called infectious pneumonia, and for a great many others to which at the present day different names are applied. I hope, however, that further researches and observations will soon complete the results already obtained, and that the pneumonias of the horse will soon admit of a scientific classification.

It seems to me that these first results, if they are well founded, ought to lead to a radical change in the prophylaxis and treatment of pneumonia. The inoculation of healthy subjects with the attenuated bacilli of influenza ought to protect them not only against influenza, but also against the pneumonia of the same nature. Vaccination ought therefore to be employed as a means of prevention in contaminated stables, while the affected animals ought to receive at the same time anti-influenza and anti-strangles serum.

NOTE ON THE EXPERIMENTAL TRANSMISSION OF WARTS IN THE DOG.

By J. M'FADYEAN and F. HOBDAY, Royal Veterinary College, London.

At a later part of this number (p. 363), our colleague, Professor Penberthy, records certain clinical facts which pointed very distinctly to contagion as the cause of the development of warts in the mouths of a number of dogs. We had ourselves for some considerable time suspected that these structures had a contagious origin, and we seized the opportunity presented by the cases in the College Infirmary to put our suspicions to the test of experiment.

Experiment I.—Our first experiment was made with a wart excised with sterile instruments from the buccal mucous membrane of one of the young fox-hounds referred to in Professor Penberthy's article. In this animal the lining membrane of the mouth was closely studded with warts, and a thick fringe of these structures was growing on the margins of the lips, as shown in the annexed figure (Fig. 1, p. 342).

13th April 1898. The cut surface of the recently excised wart was used to rub a small area of lightly scarified mucous membrane on the right upper lip of each of three dogs, viz., a pug and two fox terriers (A and B).

No effect was observable until about a month afterwards, when a slight ridge of growth began to show itself at the seat of inoculation in the fox terrier A. A few days later a similar growth could be detected on the right upper lip of fox terrier B ; and about six weeks after inoculation the pug also developed minute elevations at the scarified spot.

In the two fox terriers the papillomata continued to increase in size until about the 10th of June, at which time they had attained a size and appearance almost identical with those of the natural warts in the mouth of the fox-hound. For about a week afterwards the warts

remained of apparently the same size. They then began to shrink, and by the 28th of June they had almost entirely disappeared.

The warts on the lip of the pug grew rather more slowly; on the

FIG. 1.

Head of fox-hound, showing profuse growth of warts on the lips. Natural case.

30th of June, when the animal was destroyed by chloroform, they had the size and appearance shown in the annexed figure (Fig. 2).

FIG. 2.

Head of pug, showing warty growths at seat of inoculation on right upper lip.

Experiment II.—On the 30th of June 1898 one of the experimental warts shown in Fig. 2 was snipped off with sterile scissors, and gently rubbed against a small area of scarified mucous membrane on the

left side of the upper lip of each of the fox terriers, A and B, of the preceding experiment, and on both sides of the upper lip of a bull terrier. Another small piece of wart from the above pug was pounded in a sterile mortar with a little sterile water, and $\frac{1}{3}$ cc. of the turbid liquid thus obtained was injected under the skin of the thigh in fox terrier B and in the above bull terrier.

The subcutaneous inoculations and the attempts to re-infect the mouths of the two fox terriers had entirely negative results, but on each side of the lip of the bull terrier papillomata formed at the scarified spot. The first growth was noticed on the 2nd of August; they continued to grow till about the 26th of the same month, and by the 6th of September they had almost entirely disappeared.

Experiment III.—6th September 1898.—A typical wart (occurring naturally and the only one present) was excised with sterile instruments from the tongue of a fox terrier, and rubbed gently against a small area of scarified mucous membrane on the right side of the upper lip of a retriever pup, and on the lower lip (mesially) of the bull terrier of Experiment II. A similar attempt was made to infect the surface of the retriever pup's penis near its tip.

On the 7th of October a commencing warty growth was visible on the right side of the upper lip of the retriever pup, and it grew rapidly during the next fortnight.

On the 21st October the major part of the warty growth was snipped off with scissors, and with a portion of it a small scarified area on the opposite lip was rubbed. Since then the growth on the right side (originally inoculated) has increased very considerably in size, and at the present time (22nd December) it is several times larger than the combined warts shown in Fig. 2. Some small warts have also now formed at the place scarified and rubbed on the left side. No growth has formed on the penis, or on the lower lip of the previously infected bull terrier.

Experiment IV.—21st October 1898. A piece of the warty growth was excised from the upper lip of the retriever pup of the preceding experiment, and rubbed against a small scarified area on the mucous membrane of the left side of the upper lip of an adult retriever dog and of a sheep-dog. A small area on the penis of the retriever was treated in the same way, and another attempt was made to re-infect the bull terrier of Experiment II. on the lip. On the 7th of December it was obvious that the experiment had succeeded in the case of both the retriever and the sheep-dog, and at the present time (22nd December) there is a considerable warty growth at the scarified part of the lip in each of these animals. No growth has formed on the lip of the bull terrier, or on the penis of the retriever. ·

Both the natural and the experimentally induced growths had a typically papillomatous structure. This was already evident from their naked-eye appearance, and a microscopic examination has confirmed it. Each wart has a rather delicate vascular connective tissue in its centre, while the superficial part of the growth is everywhere made up of a stratified squamous epithelium.

The experiments therefore conclusively prove that the common papilloma of the dog's mouth is transmissible, and they support the clinical evidence in favour of contagion being the common cause of

such growths.   They also show that without any treatment whatever
such papillomata may disappear by a process of gradual shrinking
and absorption, and they suggest the thought that the credit claimed
for some methods of treatment may be undeserved.

Lastly, the experiments indicate, though, owing to their small
number, they cannot be said to prove, that after disappearance of a
first crop of papillomata the animal is left in a measure protected
against a second infection of the same kind.

EDITORIAL· ARTICLE.

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THE RELATIONSHIP BETWEEN HUMAN AND
BOVINE TUBERCULOSIS.

SINCE the identity of human and bovine tuberculosis was first
demonstrated very diverse opinions have been expressed by medical
authorities as to the frequency with which the disease is communicated
from the one species to the other.   For a number of years after Koch's
discovery of the tubercle bacillus the view that " human phthisis fre-
quently comes from the butcher's stall " was rather widely adopted,
and this opinion was in very large measure responsible for the crusade
originated against tuberculosis of cattle.   Within the last few years,
however, the view which has been steadily gaining ground among
authorities on questions of human sanitation is, that the immense
majority of cases of human tuberculosis originate in human sources
of infection, and that if any great reduction in the present prevalence
of the disease is to be brought about prophylactic measures must be
directed against the spread of the infection from man to man.   An
important contribution to this question has recently been made by
Sir Rirchard Thorne in his Harben Lectures on the Administrative
Control of Tuberculosis, and it is of interest to note the estimate
which he has formed of the dangers incurred by the public from the
consumption of tuberculous meat and milk.

At the outset Sir Richard Thorne reviewed the measures which had
been adopted during the last forty-five years in the control and
prevention of the different forms of tuberculosis, and the results
achieved as shown by certain vital statistics prepared for the Royal
Commission on Tuberculosis by Dr Tatham.   From these statistics
it appears that during the last forty years there has been a very
notable decline in the death-rate from tuberculosis among human
beings in this country.   This decline has been constant, and it
applies to all forms of tuberculosis collectively, but it has been most

marked in the case of phthisis, which at certain periods of life has experienced a reduction of 60 per cent.

These highly satisfactory results were mainly ascribed by the lecturer to administrative measures which had secured increased movement of air and greater access of light around and within human habitations, and which had led to a lowering of the level of subsoil water in our towns and in the neighbourhood of villages and dwellings. Sir Richard Thorne points out that all this had been effected before the discovery of the tubercle bacillus had led to any special action based on bacteriological knowledge, and he anticipates that if as much can be effected during the next half century death from pulmonary tuberculosis at many age periods will have been entirely abolished in this country.

It would be of immense interest to know how cattle have fared with respect to the same disease during the half century which has brought such a remarkable decline in the death-rate from human consumption. Unfortunately, there are absolutely no reliable data on which to found a comparison. At the present time many people have adopted the opinion that bovine tuberculosis is vastly more prevalent than it was half a century ago, but there does not appear to be sufficient evidence to warrant any opinion on the subject. In this matter it is very easy to drop into error (1) by forgetting that we are only now beginning to acquire an accurate notion of the number of cattle affected with tuberculosis, and (2) by overlooking the fact that death-rate and prevalence are not the same thing.

The use of tuberculin on live cattle, and rigid *post-mortem* inspection of apparently healthy cattle, appear to justify a very high estimate of the present prevalence of tuberculosis among adult bovine animals in this country—probably not less than 30 per cent., but there is no reliable information as to the number of cattle that die annually from the disease. A comparison between human and bovine tuberculosis in this respect, even if we had reliable figures, would be vitiated by the fact that tuberculosis in cattle is seldom allowed to run a natural course when it threatens to end in death. But even if we reckon with the fatal cases those in which the animal is slaughtered because of tuberculous symptoms, the death-rate from tuberculosis among cattle over the whole country is not very great.

We have said that there is no reliable information upon which one may base a comparison between the present and the past with regard to the prevalence of tuberculosis in cattle, but in a recent article on the subject[1] M. Leclainche attempts to show that the disease has spread in an alarming manner even within the last ten years. In support of this view statistics from various slaughter-houses are cited, showing, in the case of Leipzig, for example, a rise from 11·1 per cent. in 1888 to 33·02 in 1895. These figures we believe to be entirely

[1] "Revue de la tuberculose," 1898, p. 307.

fallacious as an index to the prevalence of the disease, since it is almost certain that during the period referred to the search for tuberculous lesions in public slaughter-houses has become much more severe. M. Leclainche cites this country in evidence of the spread of the disease, and says: "Great Britain has seen its finest breeds successively decimated and threatened with complete destruction." This is a very exaggerated estimate of the state of affairs; at least, if it is not, the owners of tuberculous herds in this country have been remarkably successful in concealing their losses. No doubt many of our finest herds contain a large proportion of tuberculous animals, but the fact has been discovered by the tuberculin test, and not by the death of any considerable number of the animals. In fact, in this matter we are probably no worse off than France itself. Only a few years ago, in a Bill for dealing with tuberculosis laid before the French legislature, the proportion of French cattle affected with the disease was officially estimated at 1 per cent.; but M. Leclainche now admits that the tuberculin test has revealed from 50 to 80 per cent. of affected animals in herds previously thought to be free from the disease, and he places the number of animals attacked in the whole of France at somewhere between 10 and 20 per cent.

It thus appears that we have statistics indicating the death-rate from human tuberculosis, but not the proportion of human beings attacked; while, on the other hand, we have some fairly reliable indications of the number of cattle affected with tuberculosis, but none as to the number of deaths caused by the disease. May one assume that the very gratifying reduction of the death-rate from tuberculosis among human beings has been accompanied by a corresponding decline in the number of persons attacked? It may at first appear that this question must have an affirmative answer, but further reflection makes the question more doubtful. It certainly appears to be possible that the decline in the death-rate from human phthisis may be in part, perhaps even mainly, due to a more rational and successful treatment of the clinically tuberculous. Let us suppose for a moment that this explanation of the figures is rejected, and that it is maintained that the decline in the number of persons attacked has been equal to the decline in the death-rate since 1851. We have said that statistics regarding the present prevalence of the disease are lacking, but an estimate of the number of persons attacked may be based on the number of cases in which tuberculous lesions are detected in the bodies of persons submitted to *post-mortem* examination in hospitals. A pathologist of large experience estimates that at the present time such lesions are present in about 30 per cent. of the bodies examined —a proportion which may at first sight appear incredible, but which is no more surprising than to find a like proportion of animals reacting to tuberculin in an apparently healthy herd. Hence, it would appear that, if the decline in the prevalence of the disease has kept

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pace with the diminution in the death-rate, about 50 per cent. of the inhabitants of England and Wales must have been tuberculous half a century ago.

The connection between damp soil and phthisis was first put forward by the late Sir George Buchanan, Sir Richard Thorne's predecessor at the Local Government Board. Buchanan went so far as to say that " wetness of soil is a cause of phthisis to the population living upon it." This was before the discovery of the tubercle bacillus, and before it had been recognised that tuberculosis is a purely contagious disease. If it were an established fact that draining of the soil, without any other change in the circumstances affecting the spread of phthisis, is always followed by a decline in the death-rate from that disease, we should have to recognise a relationship of cause and effect between dampness of soil and phthisis, even although we could not understand the exact way in which the dampness of soil induced the disease in persons living on it. But at the present day it cannot be admitted that the quality of the subsoil has the importance which Buchanan ascribed to it as a factor in the etiology of phthisis. A wet subsoil cannot cause phthisis without the tubercle bacillus, and when the conditions and habits of life of the population are favourable to infection the disease will spread on the dryest of subsoils. These statements are incontrovertibly true for tuberculosis of animals, and they probably hold good for human phthisis also.

Sir Richard Thorne called attention to the fact that while during the last half century there has been a remarkable decline in the death-rate from phthisis, there has not been a corresponding diminution in the number of deaths returned under the head of tabes mesenterica. At most ages the decline under that head has been quite trivial during the last half century, and there has been an actual increase in the rate of death from this form of the disease in children under one year of age. The lecturer boldly asserted that the explanation of this remarkable fact is to be found in the circumstance that phthisis is mainly due to aerial infection, and is therefore influenced by improvements in the direction of ventilation, lighting, etc., while tabes mesenterica is caused by alimentary infection through the medium of meat and milk, and is therefore unaffected by such sanitary measures. Tuberculous meat he believes to be only in a minor degree responsible for cases of tabes mesenterica in the human subject, but he considers the loss of child life from the consumption of tuberculous cow's milk to be appalling. This is a very grave indictment of the milch cow as an agent in the dissemination of tuberculosis among human beings, and if it were proved it would justify the introduction of the most energetic measures against bovine tuberculosis.

Apparently Sir Richard Thorne's charge against tuberculous

milk might be formulated somewhat as follows. During the last fifty years there has been a marked decline in the death-rate from tuberculosis due to aerial infection (phthisis). During the same period there has been only a slight decline in the death-rate at all ages from that form of tuberculosis which is ascribable to alimentary infection (tabes mesenterica), and among children under one year of age there has been a notable increase in the mortality from that form of the disease. This increase in the proportion of deaths in young children returned under the head of tabes mesenterica has been coincident with a large increase in the consumption of cow's milk as an article of human food. The use of tuberculous milk is therefore the main cause of tabes mesenterica in children.

There are several obviously weak points in this indictment. The first of these touches the application of the term tabes mesenterica by medical men in certifying the cause of death. Dr Tatham, who presented the statistics on which the charge against milk is framed, says[1]: "Tabes mesenterica is an indefinite but time-honoured term, under which are probably included, in addition to tubercular affection of the mesenteric glands, peritoneum, and intestines, a considerable but unknown number of ill-defined ailments in which wasting and diarrhœa are prominent symptoms. This disease in its fatal form affects young children almost exclusively."

Now, when it is remembered that all except an inconsiderable fraction of cases of so-called tabes mesenterica are certified without a *post-mortem* examination, it becomes impossible to repose great faith in the Registrar General's returns so far as they pretend to be a record of the deaths from alimentary tuberculosis. It cannot even be said to be improbable that the majority of the cases returned under this head have nothing whatever to do with tuberculosis. And when one examines with a little more detail the figures given in Dr Tatham's tables one finds certain variations in the incidence of the mortality from the different forms of tuberculosis that are wholly inexplicable on the view that the terms have been consistently employed throughout, have embraced all the deaths from that form of the disease, and have not included many deaths from other diseases. For example, in the decennium 1851-60 the deaths returned under the head of phthisis in children under one year of age numbered 2032 per million births, and in the period 1861-70 they had fallen to 1370 per million births, but during precisely the same period there was scarcely any decrease in the death-rate from phthisis at thirty-five to forty-five years of age (4091 per million living in 1851-60 and 4026 in 1861-70). It is very difficult to understand what were the conditions that .effected such a great reduction in the mortality from phthisis among infants between 1860 and 1870,

[1] Report of the Royal Commission on Tuberculosis, p. 357.

and yet were without any sensibly ameliorating influence on the mortality from phthisis at middle life.

The statistics relating to tabes mesenterica are no less puzzling. Take for example the following :—

Table showing Mortality from Tabes Mesenterica.

| Period. | Per Million Births. | | | | |
	Total under 1 Year.	*1 to 2 Years.*	*2 to 3 Years.*	*3 to 4 Years.*	*4 to 5 Years.*
1851-60	3169	1906	787	344	214
1861-70	3800	2140	774	315	187
1871-80	4467	2231	695	279	167
1881-85	4356	1931	638	254	164
1886-90	4462	1705	526	223	138
1891-95	4046	1504	469	208	137

From this table it would appear that during the last forty years there has been a substantial increase in the death-rate from tabes mesenterica among children under one year, and a distinct but unequal decrease from the same cause at all ages between one and five years of age. Assuming that tabes mesenterica is mainly or largely attributable to the consumption of tuberculous cow's milk, how can one explain these discrepancies? It is scarcely open to doubt that a very much larger quantity of cow's milk is consumed by children between one and two years of age, than by those under one year old, seeing that the majority of children of the common people are still suckled at the breast, and yet the mortality from tabes mesenterica has been steadily declining during the last forty years among the former, while it has increased among the latter. Furthermore, the death-rate from tabes mesenterica has declined since 1851-60 among persons from five to fifteen years of age, and increased for every later period of life.

Again, it appears from a table submitted to the Royal Commission on Tuberculosis by Dr A. K. Chalmers that in Glasgow the death-rate from tabes mesenterica has fallen from 560 per million in 1883 to 270 per million in 1894, and that this considerably exceeded the decline in the mortality from phthisis during the same period, viz., from 3361 per million in 1883 to 2271 in 1894. Now, in order to make these figures

fit in with the view that tabes mesenterica comes mainly from the milking pail, we would have to assume that there has been a great falling off in the consumption of cow's milk in Glasgow during the last twelve years, or a great decline in the number of tuberculous cows contributing to the milk supply of that city. We believe there is no evidence to lead one to suppose that either of these things has happened.

The conclusion at which we arrive is that the Registrar-General's returns under the head of tabes mesenterica cannot with confidence be accepted as an index to the proportion of human tuberculosis contracted through drinking the milk of tuberculous cows. It is almost certain that the term covers such a large number of deaths from other causes as to entirely vitiate the statistics. Strong evidence in support of this view is furnished by a chart which Dr Tatham submitted to the Royal Commission on Tuberculosis to show the annual fluctuations in the death-rate from tabes mesenterica and diarrhœa during the last twenty years. The parallelism between the two lines is very striking, and almost compels one to believe that while some practitioners return cases of death following diarrhœa and wasting under the head of diarrhœa, others return them as tabes mesenterica.

But even if one could accept all the cases returned under the head of tabes mesenterica as undoubted instances of tuberculosis affecting the abdominal organs, it would not be justifiable to maintain that all such cases had their origin in tuberculous milk. In the households of the very poor the circumstances which contribute to aerial infection with tuberculosis must also carry with them some risk of food contamination, and consequently of alimentary infection; and some customs, such as kissing, tend specially to the infection of human beings by way of the alimentary canal. Taking all these facts into consideration, we should prefer to frame the indictment against milk, not on anything disclosed by the Registrar-General's statistics, but on the ground that tuberculosis of the cow's udder is not a rare disease; that the milk of a tuberculous udder when tested by experiment on animals is invariably infective; that at the present time there is hardly any legal obstacle to the sale of such milk; and that such milk is beyond doubt being sold every day in many parts of this country. We may not be in a position to say precisely what proportion of cases of the human disease are caused by milk, but it is impossible to doubt that some cases originate in that way, and the public have surely a right to demand that if there are any reasonable measures by which this danger can be averted or minimised, they ought to be put into force without further delay.

Reviews.

Compendium der speciellen Chirurgie für Thierärzte. Von Dr Eugen
Fröhner, Professor und Dirigent der chirurgischen Klinik an der Königl.
Thierärztl. Hochschule in Berlin. Stuttgart, Ferdinand Enke 1898.

This is a very useful student's text-book of a little over 300 pages. The
preface explains that it is a summary or digest of the author's course of
lectures, which he has been in a measure compelled to publish owing to the
issue of an unauthorised version of the same. The various surgical diseases
of the domesticated animals are succinctly treated under the heads of cause,
symptoms, and treatment. The information given within small space is
wonderfully full, and, although it is mainly a student's book, it is by no
means beneath the attention of the experienced practitioner, since it gives an
accurate sketch of the present day position of scientific veterinary surgery.

The Annual Statistical and General Report of the Army Veterinary Depart-
ment for the Year ending 31st March 1898.

The Annual Report of the Director-General of the Army Veterinary
Department shows that the health of the army horses in the United
Kingdom during the past year was very satisfactory, although the rates of
inefficiency, mortality, and admissions to treatment were all somewhat higher
than during the previous twelve months. The amount of inefficiency from
disease and injuries was 65·12 per cent. of the average strength, and the
mortality 2·73 per cent., the corresponding figures for the previous year
having been 61·10 and 2·52 respectively. As compared with the previous
year, there was an increase of 77 in the admissions for diseases of the chest
and air-passages, and of 150 for strangles. No case of glanders or farcy
occurred during the year, but in consequence of some suspicion attaching to
a horse that died from "pyæmia" the two animals that stood next to this
one were tested with mallein, with the result that neither reacted.
Veterinary-Captain Blenkinsop, in his report regarding the army horses in
Egypt, records the fact that the animals belonging to the British army of
occupation were free from glanders during the year, although the disease
existed to a considerable extent in Cairo and Alexandria. Testimony is
given to the great value of testing suspected horses with mallein.
In South Africa a severe outbreak of glanders occurred amongst the local
transport at Etchowe, but by the use of mallein and the prompt slaughter of
all the horses that reacted the disease was stamped out. The rate of sickness
and mortality amongst the army horses in South Africa was unusually high
during the year.
The Report shows that, as in former years, very excellent results, at a
trifling cost, have been obtained at the Army Vaccine Institute.

Examination of Horses as to Soundness and Selection as to Purchase. By
Edward Sewell, M.R.C.V.S.L. London : Bailliere, Tindall & Cox. 1898.

ACCORDING to the preface this book has been written "to assist the horse-
owner, the farmer, and the colonist to judge for himself as to the practical
soundness of a horse he may be interested in . . . " which, coming from
a practising veterinary surgeon, is exceedingly generous, if nothing more than
that. Examining horses as to soundness is work for veterinary surgeons, and

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there is little danger of immediate invasion of the field by farmers and
colonists, or by those, more especially, who may read this book.   It is easier
to write a long bad book than a short good one.   This is an indifferent pro-
duction, and but little else could be expected from an attempt to crowd into
eighty-six small pages—including illustrations of dentition—all that should be
known by farmers and others of unsoundness in horses.   The book has many
and various faults, but only a few need be pointed out; the rest will be dis-
covered.   Every veterinary surgeon knows, or ought to know, that the examiner
who relies solely on the catoptric test of sight may overlook small cataracts.
The condition of the nasal membrane and of the submaxillary and other glands
has some importance.   The poll, throat, trachea, jugulars, and neck receive
attention from most veterinary surgeons.   The horse's *arm* does not extend
downwards from the elbow, which, by the way, is sometimes capped.   Un-
nerved horses and others affected with navicular disease are sometimes offered
for sale, but the horse buyer must judge for himself, for he will look in vain
into this book for the needful information.   Loose wall is described as false-
quarter, which is not only a different defect, but one that is incurable.   Little
is said of defects of the hindquarters, fractured ilium or ischium is not even
mentioned, and what is given concerning the stifle might have been left out
without loss.   Curb and its consequences are curiously explained, and the
existence of bone spavin is considered sufficient to bar the purchase of any
horse, notwithstanding common experience of the usefulness of spavined hocks.
In the chapter dealing with action, p. 43, mention is made of a £350 horse
" which had the most extravagant action in front, and although the horse
apparently went sound, the feet had every disease that the horse's foot could
possibly be afflicted with . . ."   This may not be an extravagant state-
ment of an exceptional case, but it reads like one.   The colonist will " judge
for himself."   The pages devoted to " examination of the respiratory apparatus "
may serve their purpose, and the illustrations of the teeth constitute about the
only distinctly commendable feature of a book which, in a sense, recalls
Christopher North's description of a sheep's head—" There is a fine lot of con-
fused eating in it."   If a second edition should be called for, the author would
be well advised to recast the whole work, fill in omissions, and endeavour to
supply less objectionable explanations of the defects of horses.

Cape of Good Hope Department of Agriculture.   Report of the Colonial
      Veterinary   Surgeon   and   the   Assistant   Veterinary   Surgeons   for   the
      year 1897.

As was to be expected, this report teems with matter of great interest regarding
the recent disastrous invasion of South Africa by rinderpest and the measures
put into operation to check the plague.   We gather from it that these measures
have by no means always given the results expected, and that in such circum-
stances matters did not always proceed quite smoothly between the Colonial
veterinary surgeon and the medical experts who were deputed to continue the
researches initiated by Professor Koch.   The report appears to us to be an
absolute vindication of the work of Mr Hutcheon and his staff of veterinary
assistants.
      It is generally known that Professor Koch, before he left South Africa in
the early part of 1897, recommended that an attempt should be made to combat
the rinderpest invasion by carrying out protective inoculation of the healthy
cattle with bile from animals that had suffered from the disease.   He did not
believe that this method would have the effect of spreading the disease, and
his last words on it were, "that this discovery is practically applicable I con-
sider absolutely proved."   Apparently inspired with confidence by this assertion,
Mr Hutcheon and his assistants immediately began to put the bile method *in*

operation. The result is indicated in the following extract from a report by Assistant-Veterinary-Surgeon Shepherd :—

"It has been freely stated that it is impossible to give rinderpest to an animal by injecting rinderpest gall. There have been over 50,000 cattle inoculated in this district by the Koch method, and, as far as my information goes, the disease appeared on every farm with but two or three exceptions about eight days after inoculation." This experience was by no means exceptional, and, indeed, nothing is more clearly proved by Mr Hutcheon's report than that the bile method of inoculation is not a means of warding off an invasion of rinderpest, but rather a method of spreading the disease. This is an illustration of how a laboratory method, theoretically perfect, may break down when it is applied practically on a large scale.

Needless to say, in face of such results, the bile method had to be generally abandoned, and Mr Hutcheon's report enables one to follow the various other methods — inoculation with antitoxic serum, inoculation with defibrinated blood, infection with virulent blood followed by injection of antitoxic serum, etc.—which have been in succession adopted. It is clear from South African experience that none of these are methods of stamping out or prevention in the proper sense of the word. They might much more correctly be described as methods of spreading the conflagration and of controlling the resulting new fires.

It is well to note this, while recognising that the methods lately pursued in South Africa have saved the lives of thousands of cattle there. Stamping out in that part of the world appears to have been impossible, and other measures had to be employed, but no greater mistake could be made than to suppose that the South African experience points a new way of dealing with rinderpest should it again appear in Western Europe. As Mr Hutcheon observes, "the only possible security against the infection of rinderpest being carried is to leave no infection to carry." Should it ever again be landed on our shores, the pole-axe and not the hypodermic syringe is the instrument with which it must be opposed.

CLINICAL ARTICLES.

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NOTES ON SCABIES IN FIELD RABBITS.

By A. M. TROTTER, M.R.C.V.S., Glasgow.

THE following notes on scabies in field rabbits may prove interesting.

This disease has been prevalent for many years on several estates in the vicinity of Glasgow, causing a large annual mortality. Many theories have been promulgated as to the causation of this plague ; of these, in-and-in-breeding, overstocking, and dirty pastures have appealed most to the initiated. An attempt was made by one proprietor some years ago to eradicate it by introducing a number of tame rabbits, but the effort proved abortive. In another instance, acting on the assumption that overstocking was the cause, slaughter was resorted to, but this proved equally unsuccessful.

Rabbits of all ages may be found affected. Gerlach has never seen it extend beyond the face, but Railliet records instances where it has spread to the neck, the lower half of the auricular concha, the fore-legs to the elbow and the hind legs to the hock. In this outbreak

no portion of the body was exempt from its invasion. Commencing, as a rule, in the region of the head or the pubes, it gradually spread and involved the adjacent tissues. In very advanced cases, head, outer surface of the auricular concha, neck, legs below elbow and hock, pubes, and body, were frequently found affected in the same subject to such a degree that prehension, respiration, vision, and locomotion were more or less interfered with. When thus affected it was associated with extreme emaciation. In the early stages the hair of the invaded part falls off, and close examination reveals a thin crust, which gradually increases in thickness as the disease progresses. These crusts are of a dirty greyish colour, and their surface is intersected by deep fissures. On manipulation they are found to be dense and pultaceous in character, and can only be removed with difficulty, exposing a raw bleeding surface. If a small quantity from the deeper part of the crust be taken, macerated in methylated spirits for a short time, and examined under a low power of the microscope, numerous acari are seen along with their excrement, ova, hair, and epidermic cells. There is nothing of note to be recorded regarding the parasites, which belong to the variety known as sarcoptes minor, var. cuniculi.

Gerlach affirmed that the sarcopt of the rabbit would not live on any of the domestic animals, not even on the cat.

SEPARATION OF UPPER EPIPHYSES OF BOTH HUMERI IN A MARE.

By H. THACKERAY, Student, Royal Veterinary College, London.

THIS remarkable case occurred in Messrs Hurrell's practice at Southminster in the spring of this year, the subject being a three-year-old cart mare.

The animal had been in the possession of the owner, a small farmer, for about ten days, having been bought by him from a travelling hawker, so that her previous history could not be ascertained. She was apparently quite sound, and since her purchase had been worked regularly at light ploughing and harrowing. She did her work well, and never evinced any sign of lameness. On the day that this happened she was turned out in a small croft adjoining the farm buildings, and at mid-day was seen by the waggoner to be quietly feeding; half an hour afterwards she was found down in the middle of the croft, and all attempts to get her up were fruitless.

Messrs Hurrell were sent for, and on arriving the mare was found to be lying on her near side. There were no external signs of injury beyond considerable swelling at the point of the shoulder, but examination and manipulation revealed an apparent fracture of the upper end of the right humerus. She was put on a gate, and dragged into an open shed where it was proposed to sling her, but on turning her over it was found that an exactly similar state of affairs existed on the near side, and there was an apparent fracture of the upper end of the left humerus also.

On this it was decided to kill the animal, and on making a *post-mortem* examination it was found that there was separation of the upper epiphyses of both humeri from the shafts, the sharp ends of which had underrun the muscles at the posterior borders of the scapulæ, forming pockets about 6 inches deep, and these contained granular masses of coagulated blood mixed with small spicules of bone. The epiphyses were in their normal positions, the articular surface of each being in apposition with the glenoid of the scapula, and the capsular ligament intact.

There was nothing to show what caused this sudden simultaneous separation ; a careful examination of the croft failed to show any sign of the mare having been galloping or jumping, there was nothing she could have come in contact with that would have caused such an injury, and she was found close to where she was last seen feeding.

In the accompanying figure (p. 356), B B show the separated epiphyses viewed from below. In each case the epiphyses appears to have carried with it a thin plate of bone from the shaft.

FRACTURE OF THE TIBIA, WITH LATE DISPLACE-MENT OF THE FRAGMENTS.

By J. MACQUEEN, F.R.C.V.S., Royal Veterinary College, London.

A GREY harness mare was kicked on the inside of the left hind leg on 28th August. She was sent out next day, and continued at work every day until 19th September, when she was rested in consequence of lameness and swelling of the injured leg. Fomentations and liniments were applied, but as no improvement resulted from this treatment the mare was sent in a conveyance to the College on 10th October. She was then very lame of the near hind leg, which was swollen and very painful on the inside over a surface extending nine inches above the hock. There was a small wound without discharge, and considerable œdema. Temperature and pulse normal ; appetite good.

No history of the accident was forthcoming at that time, but the symptoms suggested severe injury—probably fracture of the tibia. The mare was placed in slings, and the owner was informed of the nature of the case. As the mare was a favourite, the owner refused to have her slaughtered. Treatment failed to effect any change for the better ; and on 25th October, as the opposite hind leg was much swollen and very weak, the mare was removed from the slings. For two days she did not lie down. On the 28th October she was found standing as usual, but with bedding marks on her quarters. She lay down in the afternoon, and at night the injured leg was found bent in an abnormal direction. She was promptly killed.

In the accompanying figure, A reproduces a photograph of the fractured bone. A large callus had formed on the inner aspect of the

lower half of the bone, but the edges of the fragments are quite sharp and show no evidence of reaction at the upper part of the fracture. Apparently the kick received on the 28th of August had bruised and
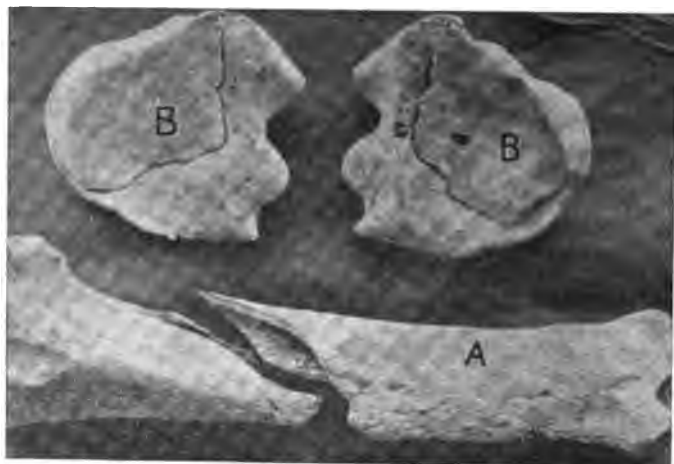
A. Fracture of tibia (Professor Macqueen's case).
B B. Separated upper epiphyses from the humeri (Mr Thackeray's case).

fissured the shaft of the bone at the seat of impact, and this partial fracture had been completed by upward extension of the fissures after the animal was taken out of the slings, or when she lay down on the 28th of October.

TRAUMATIC DISEASE OF THE STIFLE JOINT.

By J. MACQUEEN, F.R.C.V.S., Royal Veterinary College, London.

ON the 7th May 1898, I received from Mr Harry Olver, F.R.C.V.S., Tamworth, the stifle of a young cart horse, with the following particulars:—"About two months ago the horse was kicked on the off stifle, which a few days afterwards discharged synovia freely. The horse was placed in slings and treated, and the discharge ceased. A good deal of thickening remained, and no weight was ever put on the affected leg. A fortnight ago my opinion was asked concerning the case, and I thought there was no chance of a useful recovery. The horse was destroyed, and on seeing the interior of the joint I was struck with its condition. The entire absence of cartilage from a considerable portion of the articular surface appeared to me extra-ordinary, as the patient did not seem in excessive pain, but fed well and rested comfortably in slings. Thinking the case rather an exceptional one I requested that the joint be sent to you. It may interest both you and your class."

The condition of the bones of the stifle joint is shown in the accompanying figure. The outer division of the femoro-tibial joint was almost normal, but there was extensive loss of articular cartilage

and erosin of the subjacent bone in the femoro-patellar, and inner division of the femoro-tibial joint. Around the articular surfaces

Diseased stifle joint.

there was an abundant growth of newly formed nodules and spicules of spongy bone.

THE CONTAGIOUSNESS AND FATALITY OF GLANDERS.

By H. H. WHITLAMSMITH, M.R.C.V.S., London.

AT a meeting of the Central Veterinary Medical Society, London, during a discussion on the use of mallein, Professor M'Fadyean expressed the opinion that "Glanders is not so contagious or fatal as we have been accustomed to think." The following observations appear to support that view :—

In March 1895 there was an outbreak of glanders in a stud of sixty cart horses in South London, and since then I have had the whole of these animals under close observation. They are stabled at two depôts a mile apart—forty-five at one yard and fifteen at the other. The stables are well drained and ventilated, and the horses well fed and regularly worked.

In 1895 all clinically affected animals were slaughtered, and the remainder tested with mallein. Eight horses reacted strongly, with high temperatures and large swellings at the seat of inoculation.

These reacting animals were placed in a range of stabling by themselves, and they have been worked to the present time, when they are looking well and healthy, with the exception of two that were killed because of being past work (glanderous nodules in lungs on *post-mortem*). The above were all tested with mallein again in April 1898, and reacted. The temperature rose as high as formerly—104°-105° F., but the swelling on the neck was only about half the size, and not so painful, and the horses did not stop feeding as they did in 1895.

These horses come into close contact daily with the healthy ones while waiting in the yards, often touching each other during the time their vans are being loaded, but only in the open air.

In 1896, owing to want of space in the healthy stables, a twelve-year-old gelding was placed in the range of stabling with the affected horses. He is in a box separated from the others by wooden partitions, and has been regularly worked in a team with the two animals that gave the biggest reaction to mallein in 1895. No precautions have been taken in his case as regards separate brushes, pails, etc. He was inoculated in April 1898, and gave not the slightest reaction.

In April 1898, in the yard where fifteen horses are stabled, we had a case of cutaneous glanders in a seven-year-old gelding which had been brought into the stud eighteen months previously, and was supposed to have come from Lancashire, and *had never done well*. He had been stabled in a three-stall stable with two companions, and they had formed one team, and always worked together since his entry to the yard. He was, of course, killed, and the stud tested with mallein. His two companions gave no reaction.

In the next stable, four-stalled, separated from the former by brick walls, one animal reacted—a ten-year-old roan gelding. He had been in this stable a year. His three companions gave no reaction.

Table of roan gelding's reaction :—

Time.	*Temperature.*	*Local reaction.*
8 P.M.	$100\frac{2}{5}°$ F.	(Time of inoculation).
11 A.M.	$104\frac{3}{5}°$ F.	Large as five shilling piece, not raised.
6 P.M.	$104\frac{1}{5}°$ F.	Large as hen's egg. Horse not feeding.

On the second day temperature 103° F., swelling as large as a cheese plate, much raised and very painful. On third day temperature $102\frac{1}{2}°$ F., swelling as large as a dinner plate. Afterwards this swelling decreased in size, and the temperature gradually fell. A noticeable feature in this case was that as the temperature decreased the swelling at the seat of inoculation increased in size to a large extent. A month later this horse was again inoculated, and gave a very decided reaction.

The remaining horses at this yard gave no reaction.

On testing the forty-five horses in the large yard none reacted to mallein.

All the healthy horses are continually coming in close contact with affected ones, but only in the open air; and they also mix with horses from all parts of London, many of them old and not very valuable, and, on the law of averages, some affected with glanders, and yet we

have not had a single case of glanders in this stud of forty-five horses —that is if the mallein test is to be believed, and I think it is.

We have had two cases in the stud of fifteen horses—one animal probably being affected before coming into the stables; the other, not clinically affected, has contracted the disease during the last three years, we cannot say how, when, or where.

All the men in this firm in charge of teams have been provided with water pails to take out with them, and have strict instructions not to use the public water troughs, but I have repeatedly seen them doing so.

I think that we must believe that before an animal contracts glanders or any infective disease he must be in that state of health which allows the bacillus to freely do its work; there must be either permanent or temporary predisposition. By permanent predisposition I mean some natural constitutional weakness; by temporary, some lowering of the tone or vitality of the system, such as may be caused by disease, insufficient or bad food, extreme cold, excessive work.

In all cases the mallein used was that made by Professor M'Fadyean at the Royal Veterinary College Laboratory.

I have no doubt that the seven-year-old gelding, the subject of cutaneous glanders, was affected with the disease when purchased, which emphasises the advisability of testing all new purchases, however young or apparently healthy, and although sent from the country. It is quite as, if not much more, advisable that all imported horses should be placed in quarantine three days and tested with mallein. A case I had the other day emphasises the necessity of this testing of imported horses. I was called in to see a recently purchased Canadian horse. He had done no work at all in London, and I found him to be suffering from acute pneumonia. A week after this cutaneous glanders developed very slowly in different parts of the body. The pneumonia is gradually subsiding, and I have had him isolated and shall see how the case develops. He was brought into a stable where there has never been a case of glanders.

We have been told a great deal about the bacillus of glanders, *post-mortem* appearances, and mallein, but how little of the natural mode of infection or contagion and period of incubation!

One cannot be certain of giving the disease to a healthy animal by the inoculation of discharge from an *open* farcy ulcer, or by inoculation of glanderous nasal discharge; and, as we are told the transmission of the disease does not take place by inhalation of the expired air of diseased animals, it is difficult to see how infection does take place, unless by inspiration or ingestion of dried particles of nasal discharge, or that from cutaneous ulcers containing the bacillus or its spores.

EPITHELIOMA OF FACE AND LOWER JAW
IN A HORSE.[1]

By JNO. B. WOLSTENHOLME, F.R.C.V.S., F.R.M.S., Manchester.

WHEN making a *post-mortem* of another case, my attention was drawn by the slaughterer to a diseased face in a horse he had recently killed.

The subject was a large cart gelding about ten years of age. A fungating tumour mass about 5 inches in diameter occupied the right side of the face ; it extended up to and had destroyed the eye.

On examination of the lower jaw on the same side of the body, it was noticed that the posterior part of the ramus was somewhat enlarged, soft, and mobile. On cutting into this part no trace of muscular tissue or of bone was present; simply a soft grey adenoid material, with numerous fibrous trabeculæ.

The diameter of the circular opening in the face is from $4\frac{3}{4}$ to 5 inches in width, and involves the nasal, lachrymal, malar, and superior maxillary bones. The turbinated bones, the septum nasi, and other structures are involved in the deeper parts.

The posterior part of the ramus of the jaw had been absorbed over an area 5 inches in a perpendicular direction, and $2\frac{1}{2}$ to 3 inches in the horizontal plane.

The new growth extended downwards from a little below the condyle, and had invaded the external pterygoid, the internal pterygoid, the masseter muscles, and adjacent tissues.

This diseased portion of the jaw was scarcely noticeable as the head lay on the ground, for the new growth had occurred as an infiltration and had taken the same shape as the former tissues.

Examination of sections with the microscope showed that the new growth was that of a squamous epithelioma ; that is to say, in the lower jaw, areas of large squamous epithelial cells were found surrounded by delicate bands of newly formed fibrous tissue, in which were many small cells of granulation tissue. These areas when seen in cross section were more or less rounded, but appeared as processes or digitations where they had been sectioned along their long axis.

In places narrow tendinous bands were seen, the remains of inter-muscular tendons, or fibrous attachments. In other parts a few fibres of muscular tissue with their transverse striæ might be made out. Between these fibres lines of the epithelial growth were insinuated, which on being traced a very short distance were seen to have caused the fibres to become twisted and broken ; the striæ were lost, the fibrils swollen, and, in fact, all the distinguishing characteristics were destroyed.

In certain parts the new growth itself had undergone degenerative changes and had lost its cellular appearance.

An examination of sections from the free edge of the tumour at the orbit showed similar carcinomatous elements, but without trace of muscular tissue. There was, however, an increase in the amount of small-celled granulation tissue, together with considerable areas of extravasated blood.

[1] Specimen with sections under microscope shown at the Lancashire Meeting, 1st December 1898.

SOME CRYPTORCHID CASES.

By FREDERICK HOBDAY, F.R.C.V.S., Royal Veterinary College, London.

THE methods of operating upon cryptorchids are various, and have been described several times during the past few years in the professional journals; now that the principles of antiseptic surgery have become so generally adopted, the operation is one that can be performed with every reasonable hope of a successful termination. The following eight cases have been operated upon since March, three of them in the Free Outpatients' Clinique, and the remainder after consultation with various practitioners; all of them made successful recoveries.

In one case both testicles were in the abdominal cavity, and in another one testicle was found there; in the remainder the organs were found in some part of the inguinal canal. As is usual in such cases, the descended testicle was, in each instance, abnormally large.

The method of operating consisted in casting and securing the animal with ropes, as for ordinary castration; the patient was then placed on its back, the hind legs being held down and stretched widely apart by a rope or piece of webbing attached just below one hock and secured tightly to the other hock after passing underneath the loins. After carefully washing and disinfection of the penis and surrounding parts, the skin of the scrotum was incised in a situation very similar to that made when performing ordinary castration, care being taken not to cut any of the underlying tissues; the latter were then bored or torn through with the fingers, the object being to damage them as little as possible and yet to reach the inguinal canal. If the testis is present in this situation it can be felt with the ends of the fingers, the object now being to get a sufficiently good grip to pull it into such a position that the écraseur chain can be passed round it. Its removal is then a simple matter. The torson forceps were used in one of the undermentioned cases, but the écraseur in all the others.

In the cases in which the testis was not in the canal, the second and third fingers were cautiously passed into the abdominal cavity, and an exploration made around the internal abdominal ring. In Case 8 this yielded a successful result, a body like a thick cord being touched; upon being traced up for a short distance, the testicle came into my fingers. If this proved unsuccessful (as in Case 4) the whole hand was introduced, and a direct search made for the missing organ. After-treatment in each case consisted in that usually applied to severe wounds; where considered necessary, sutures of strong silk or wire were applied, these being removed about twenty-four hours later. The animals were turned into good, comfortable, roomy boxes, and plenty of cold irrigation and antiseptic lotion were used. The periods of convalescence averaged about ten days or a fortnight. I should have mentioned that all instruments, silk, wire, etc., were carefully sterilised by boiling, and that great attention was paid to cleanliness of hands, nails, etc.; in fact, everything was done as far as possible to minimise chances of infection from these sources.

Chloroform was not always used, but the relaxation of muscular tissue caused by this agent certainly gives a great advantage when the testicle is very high up or the cord abnormally short.

Case 1.—March. This animal, a well-built cob, had the left testicle situated in the inguinal canal, and no great difficulty was experienced in securing and removing it. The age I unfortunately omitted to note at the time.

Case 2.—June. A well-bred hunter, two and a half years old. Neither of the testicles had ever been seen in the scrotum, and when the animal was cast nothing was visible, nor could the presence of testes in the canal be demonstrated by a manual examination. In each case they were situated at the upper extremity of the inguinal canal, and, after some manipulation, were removed with the écraseur. With the exception of colicky pains the next morning, the patient made an uninterrupted recovery.

Case 3.—July. A pony with the right testicle hidden. This proved to be high up in the inguinal canal. The animal walked home (a distance of about 5 miles) immediately after the operation.

Case 4.—July. A cart colt, three years old, with both testicles undescended. Neither of them had ever been seen in the scrotum, and a manual examination from the exterior, when the animal was cast, revealed no sign of their presence. The right testicle was situated at the upper extremity of the inguinal canal, almost in the abdomen ; whilst the left one was in the abdominal cavity itself, floating about amongst the intestines. In order to find it, my right hand and wrist were fully introduced into the abdomen, and, after groping about for a few moments in the situation where I expected it to be, the testicle suddenly appeared to jump into my hand during one of the struggles made by the animal. Pressure upon it caused renewed struggling, and, after some little manipulation, it was withdrawn. This testicle was much larger than the other which had been imprisoned in the canal. The animal was, as might have been expected, very stiff and sore for a few days, the surrounding parts being much swollen, but otherwise no noticeable symptom for uneasiness occurred.

Case 5.—July. A cart colt, three years old, with the left testicle in the inguinal canal. The veterinary surgeon in whose practice this case occurred said that this testicle had at times been seen, but that it was always withdrawn out of sight and out of reach when the scrotum was touched. The case was not a difficult one, being completed without chloroform.

Case 6.—October. Bay cart colt, two years old, with the right testicle situated high up in the canal. This animal was put in a loosebox for twenty-four hours after the operation, and walked home (a distance of 5 miles) the next day.

Case 7.—November. A cart colt, three years old, with the left testicle (a very small one) situated at the extreme top of the canal. This was removed without chloroform. The wound was sutured with wire, the sutures being taken out the next day.

Case 8.—November. Cart horse, three years old, with the right testicle in the abdominal cavity. In this case, however, I did not have to introduce the whole of my hand, being able to reach the testicle with the second and third fingers. With the exception of

some disturbed breathing about four or five hours afterwards, the animal showed no untoward symptoms. Wire sutures were used, as in Case 7, and removed on the succeeding day.

CONTAGIOUS WARTY TUMOURS IN DOGS.

By J. PENBERTHY, F.R.C.V.S., Royal Veterinary College, London.

ON 29th March 1898 I received a communication from Mr Peter Irving, M.R.C.V.S., of Chipping Norton, concerning a foxhound "affected with fungous growths on the lips, roof of mouth, and tongue, extending as far as can be seen towards the larynx." The tumours were so numerous that Mr Irving deemed the case almost hopeless. Having had previous experience with a similar condition, I wrote advising that the disease should be treated as contagious, and suggesting that the dog in question should be sent to the College for inspection and treatment. The owner, Mr Albert Brassey, M.P., M.F.H., readily consented, and on 2nd April two hound puppies, a dog and a bitch, were admitted to the Royal Veterinary College Infirmary. On examination the dog showed all signs of good general health and condition. The lips, tongue (particularly the dorsum), palate, and pharynx were literally studded with warts. The growths were mostly discrete and fairly uniform in size and shape, perhaps smaller and more numerous on the lips, rather paler than the buccal mucous membrane, and apparently covered by a dense layer of epithelium. There was no hæmorrhage or sign of ulceration. On the skin of both cheeks were a few similar growths. The bitch appeared to be recovering from distemper and was rather thin, otherwise her general health seemed satisfactory. Similar growths, though not quite so numerous, existed in the same situations as in the dog, and in addition one was present on the right flank.

Both animals were then subjected to the same treatment, consisting in the internal administration of two-and-a-half grains of iodide of potassium in milk daily, and gargling mouth and pharynx with a solution of boracic acid, five grains to the ounce of water, twice daily. Every other day the warts were painted with a solution of iodine one part, water six parts. On the 5th of April some of the warts on the lips were snipped off with dressing scissors, but on account of profuse hæmorrhage this could not be far proceeded with.

On the 6th of April I received telegrams from Mr Brassey and Mr Irving stating that four more hounds were affected, and asking me to visit the kennels at Chipping Norton on the following day. Having made arrangements to leave on that day for Madrid, I replied that this was impossible, and advised carrying out the treatment on the lines originally suggested. On my return from Spain on the 19th of April I found the dog in much the same condition as when I last saw him on the 6th of April, though I received the impression that the warts were not quite so large. I learnt that for some days preceding the dog had received internally ten grain doses of calcined magnesia, a drug extolled by some Continental writers. This was immediately discontinued and the original treatment again

resorted to. The bitch had now recovered her general health, and her tumours were appreciably smaller. The treatment was continued till 20th May, the warts in dog and bitch gradually disappearing in much the same manner as similar growths on the human hand are usually observed to do. By the first week in June all traces of their existence had disappeared, and it is interesting and possibly instructive to note that at the Puppy Show in August our patients took first prize for the "best couple."

Though clinical observation, especially of a similar set of cases met with two years previously, had convinced me of the contagious nature of these growths, and led me to write immediately to Mr Irving suggesting treatment as of a contagious affection, I felt the desirability of testing the effect of inoculation, but between the date of admission and my departure for Madrid my endeavours to find a suitable subject for the experiment were unsuccessful.

Twenty couple of hound puppies, dogs and bitches, the majority of which had been "walked" singly or in couples by different individuals in the Heythrop Country, were kennelled together, and separate from the bitch and dog packs of working hounds. The first affected were the dog and bitch sent to the College on 2nd April. The huntsman had observed warts in their mouths, and, separating them from the other puppies, had been treating them a fortnight before Messrs Irving and Stephenson were consulted on 29th March. It is worthy of special remark that these two puppies had been "walked" by the same individual at the same place, and had been two months at the kennels before anything was noticed. Mr Irving writes to say that previous to hearing from me on 31st March no treatment was adopted. On the 6th of April four more cases were reported.

On the 18th of April it was reported that nine-and-a-half couples had been affected ; on the 25th April ten-and-a-half couples ; on the 8th of May thirteen-and-a-half couples ; and on the 17th of June eighteen-and-a-half couples. Though in some patients the warts were much more numerous than in others, in no case were they so numerous as in the first two, while in the latter only a few appeared. Three puppies only of the twenty couples escaped. The general health of all remained throughout apparently good. The disease was confined to the puppies in the one kennel. As far as I can gather, nothing of the kind had ever before been observed in the kennels of this hunt.

The mouths of the puppies were examined daily, with due observance of strict precautions against contagion, and, on the earliest appearance of any suspicious symptom, placed under the treatment before described. Mr Irving wrote me, saying: "Some of the earliest cases were very bad, others that were treated directly it was noticed had it but slightly." On my visit to the kennels on 26th April I found the cases then existing of precisely the same nature, but the growths much less profuse and generally smaller, while nothing could be more admirable than the arrangement for the treatment of the healthy and affected.

It has frequently been observed that similar growths, especially on the human hand, disappear without any treatment being specially directed to them. It is very difficult in these cases under consideration to say whether or not their disappearance was in any way related to the treatment adopted. It may be that the growths

would have disappeared spontaneously. It may be that some circumstances, intrinsic (as advancing age), or extrinsic, acting on the animals will account for the comparative mildness of the later cases.[1]

Irrespective of recent clinical and experimental investigations, the idea of the contagious or infective quality of warty growths cannot be entirely new. It was a common saying among schoolboys in my early days, "if you make the warts on your hands bleed you will be sure to have warts where the blood goes." The etiology of neoplasms is a subject of great interest and importance. Clinical observations such as the foregoing contribute something to the view that some at least possess infective properties; and this is amply verified by the results of the experiments contributed by Professors M'Fadyean and Hobday (see p. 341).

Abstracts.

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THE RINDERPEST IN SOUTH AFRICA.[2]

PROFESSOR KOCH proved that the serum of cattle which had recovered from an attack of rinderpest, when injected under the skin of healthy cattle, protected them from the poison of rinderpest for a short time. He also proved that a mixture of 99 parts of this serum with one part of rinderpest blood, when injected in a series of doses of 20 cc. into healthy cattle, gradually increased this immunity to a very great extent; but on discovering the immunising power of bile he abandoned further work on the serum treatment, as it appeared too troublesome when it came to be applied to large numbers of cattle, and also at that time to be inferior to the bile treatment on account of the impossibility of obtaining sufficient "salted" cattle from which to obtain a supply of serum equal to the demand. The disease was spreading at such an alarming rate that it was imperative that some form of treatment should be applied capable of keeping pace with the spread of the infection. Consequently, although the bile treatment was expensive from the number of cattle required to be sacrificed for its material, it was necessary to continue it for the time being, and there is now no doubt that it was instrumental in saving many thousands of head of cattle, and at the same time in checking the progress of the disease into uninfected areas to a very considerable extent. As time wore on it was found that cattle which had been rendered immune with bile after periods varying from three to six months lost their resisting power, and in many instances succumbed to fresh attacks of the disease.

Whilst this race between the spread of the infection and the attempts, more or less successful, to get ahead of it with bile inoculation was going on, Dr Kolle and Dr Turner were persevering in their scientific experiments to bring the serum treatment to perfection. Cattle which had recovered from the disease were gradually collected, and a stock of antitoxic serum was in this

[1] Pertaining to this natural tendency of the growths to disappear, as well as regarding the existence elsewhere of the same or similar conditions among puppies, it may be interesting to remark that the late Huntsman of the Heythrop, who, in forty years' experience among hounds, had never met with anything of the kind before, on visiting a friend in the Vale of White Horse, found "the same thing amongst their puppies." He states "they do not attach much importance to it, but simply wash their mouths with soda water." This coincides with my former and present experience that it is most commonly observed in puppies.

[2] From an article by Dr Maberly in the *Lancet*, 5th November 1898.

way secured. From July to the end of October, 1897, antitoxic serum in doses varying from 20 cc. to 100 cc. was used for treating herds which had already become infected with rinderpest, it being found that bile (as had been pointed out by Koch at the offstart), was quite useless if the animals treated had already been infected with disease, although they showed no apparent symptoms except a slight rise of temperature, and even animals which became infected any time before six days after the injection of bile were not safe. The serum, if given in sufficient quantity, however, proved capable of over-coming the virulence of the disease provided it was applied within three days after the initial rise of temperature. After that stage it appeared useless even in very large doses. It now slowly became certain that the only permanent immunity was to be found among those cattle which had either recovered from the disease naturally or with the assistance of antitoxic serum, and the attention of those engaged in scientific research was turned towards endea-vouring to discover some method by which a modified form of the disease could be communicated to healthy cattle, since in this way only did it appear possible that the disease would be finally stamped out. The French *savants* in the Transvaal, Dr Danysz and Dr Bordet, first proposed a method of first injecting healthy animals with immunising serum, and then keeping them continually exposed to the infection of rinderpest by either occasionally smearing their nostrils with rinderpest blood or keeping them among infected herds. In this way they hoped that the animals would take the diseases as soon as the temporary immunity produced commenced to pass off, but whilst some of the resisting power of the antitoxin was still active enough to modify the disease sufficiently to prevent a fatal result. The results, however, were uncertain, and the method was abandoned as too cumbersome for actual application. Dr Kolle and Dr Turner tried the experiment of first injecting serum and then rinderpest blood a few days after. This was followed by experiments in which rinderpest blood was first injected, followed by antitoxic serum in the course of one or two days, but the results of both these methods were uncertain, and they were also open to the objection of requiring heavy doses of serum, and the necessity of catching and casting or otherwise hand-ling the cattle treated at least twice was another drawback in actual practice. They then struck upon the happy idea of simultaneously injecting rinderpest blood and antitoxic serum into the same animal at spots in the body suffi-ciently apart to prevent the two fluids from mixing together at once. The experiments carried on at the Kimberley station in this method proved eminently satisfactory, and it was decided to give the method a thorough trial by placing at Dr Turner's disposal the cattle living on Robben Island, the Government leper station, situated in Table Bay, which up to this time were entirely free from the disease.

Dr Turner arrived at Robben Island on 1st December 1897. The first thing done was to collect all the cattle, and as several of the cows, twenty-nine in all, were heavy with calf, Dr Turner deemed it inadvisable to subject these animals to rinderpest even in a modified form, because the fever is liable to cause the cows to miscarry, and such miscarriages are frequently fatal. As it would have been manifestly imprudent to introduce rinderpest into the island while some of the cattle were unprotected, it was decided to protect these cows in calf by the injection of 150 cc. of protective serum ; this quantity had been found sufficient to immunise for six weeks or more. In this way they would incur no danger of infection when the other animals were infected. This treatment had the further advantage of demonstrating the possibility of producing a temporary immunity amongst dairy cattle without stopping the secretion of milk—a matter of very considerable importance. About five days after the injection of protective serum one of the cows so treated calved in the ordinary course, and the calf was born alive and healthy, thus showing

that even so large a dose as 150 cc. of antitoxic serum was not detrimental to life in utero. On 8th December blood which had been taken from an animal suffering from rinderpest at Kimberley on 6th December was brought to Robben Island, and Dr Turner immediately commenced his work of simultaneous inoculation on the rest of the cattle on the island—in all fifty-two in number. Each head was injected behind one shoulder with 0·5 cc. of rinderpest blood, and with an average of 25 cc. of antitoxic serum behind the other shoulder. The dose of antitoxic serum was varied according to the size of the animal, 25 cc. being considered the proper dose for animals weighing about 600 lbs. About two days after the inoculation had been performed, Dr Turner received a letter from Dr Kolle stating that the dose of this sample of serum should have been taken at an average of 30 cc. instead of 25 cc. as at first stated: consequently it was decided to give twenty-five of the animals a second larger dose of serum in order to prevent the possibility of too great a fatality. The rest were allowed to take their chance. Of these twenty-five cattle eight showed no signs of fever, chiefly owing to the second dose of serum having been too large. Of the remaining twenty-seven, which had not received a second dose of serum, four showed no signs of fever. Out of the whole number treated only one died—an old cow in very low condition; fourteen of the animals operated on were cows giving milk at the time. The secretion during the height of the fever was diminished but not otherwise altered; in one it was entirely arrested for twenty-four hours but again reappeared. Dr Turner, in order to test the quality of the fluid, drank a pint of raw milk—taken from a cow suffering from the modified disease— every morning fasting. This did not produce the slightest ill effect upon him, and the milk was used in the public institutions just as usual. As a rule, beyond fever nothing indicative of rinderpest could be noticed in the animals. The exceptions were: four purged and had discharge from the eyes and nose (one of these passed blood in the fæces), two had runnings from the eyes and nose, and four did not eat well for a day or two. The fever commenced about the sixth day after the inoculation, and lasted on an average four and a half days; the highest temperature reached was 107° F.

A second series of inoculations carried on under similar conditions at the Government farm, Tokai, near Cape Town, gave similar results. Thirty-nine animals were "simultaneously inoculated"; no deaths occurred, but twenty-nine showed symptoms of the disease. One cow gave birth at full time to a dead calf, and another aborted. The ten which showed no signs of fever were tested ten days after the first inoculation by injections of 10 cc. of virulent blood, enough to kill 10,000 full grown oxen. The result was that they reacted in just the same way as other salted cattle, and in no case were any symptoms visible. This question of the duration of immunity after an apparently unsuccessful simultaneous inoculation was further tested at Kimberley, and it was found that the immunity conferred was active for some months, and up to the present sufficient time has not elapsed to decide how long this immunity lasts, whether, in fact, it is not a permanent one. The success of this method of inoculation having been thus definitely proved, it has since superseded the other methods in the treatment of uninfected herds, and has been carried out in some instances on a very large scale. In Matabeleland, for instance, 3141 cattle were inoculated in this manner, with the splendid result that only twenty-five died, something less than 1 per cent.

According to the Cape Government statistics, published in May 1898, the total number of cattle in the colony, excluding the Transkei, before the outbreak of rinderpest was 1,639,435. Of these, 575,864 were lost through rinderpest, giving a percentage of a little over 35. The number remaining is 1,063,571, or rather over 64½ per cent. If we now apply the figures worked out in the English outbreak, and allow a percentage of recoveries of 20, which is far in excess of what actually occurred in the district of Mafeking before

Koch's discovery of the bile treatment—where we find that, out of an approxi-
mate total of 90,400 cattle, 80,000 died, 78co were shot, and only 2600
remained alive, equivalent to something less than 3 per cent.—we arrive at
some idea of the difference in results under the older method and that
brought about by the discoveries of Koch, Kolle, and Turner. Instead
of 1,063,571 head of cattle remaining, we should only have 327,887
head of cattle in the country; which means that a loss of 735,684 head
of cattle has been prevented in spite of the disadvantages of having to
carry out the scientific researches which led up to this result during the
time that the epidemic was raging. If now we estimate the cattle thus saved
at the rate of £4 per head, we arrive at the conclusion that the steps taken
to discover a remedy for the disease have resulted in a saving of £2,942,736,
or close on £3,000,000 sterling, so that even if the rinderpest bill comes to
something like two millions of money, we are still one million to the good.
Besides the fact that we now have in our possession a remedy which has
brought about the astounding result that the death-rate of the disease has
been reduced from one of between 80 and 90 per cent. to the trifling one of
something like 2 per cent., it must be borne in mind that the figures given
as representing the probable saving brought about by the scientific work done
for the disease in South Africa is reckoned at the lowest possible figure, both
as regards the probable percentage of cattle which would have been lost and
also the value per head. The amount really saved would probably be quite
fairly represented at an average of from £5 to £6 per head, and in this case
would be nearer five than three millions of money.

The facts brought to light by the scientific research carried on during the
outbreak of rinderpest in South Africa of the last two years are of so valuable
a nature that this paper would not be complete without giving a summary of
the more important of them.

A.—As regards the etiology of the disease, neither Koch, Kolle, nor Turner
have so far succeeded in satisfying themselves that they have discovered and
isolated the specific microbe of rinderpest, and their experience leads them
to believe that up to the present it has never been isolated, although various
observers claim to have done so, but on very insufficient grounds. They
believe, however, that a specific micro-organism does exist, and that it is of
sufficient size to be visible through the medium of modern lenses for the
following reason—viz., that if defibrinated rinderpest blood be filtered through
a Pasteur bougie, the clear filtered serum may be injected into clean animals
without any ill effect, while the corpuscles and fluid which remain in the filter
cause the disease. The same thing happens with the still more porous Berke-
feldt filter. Evidently the organism is either too large to pass through the
pores of the filter, or it is contained in the blood corpuscles, which are them-
selves too large to pass. To test this Kolle and Turner mixed defibrinated
rinderpest blood with five times its volume of o·2 per cent. salt solution; this
destroyed the red corpuscles. The mixture was filtered through a Pasteur
filter, and though the original fluid was virulent, the filtered solution failed in
all cases to infect animals when inoculated.

B.—An interesting question has been raised as to how gall taken from
rinderpest animals acts as a temporary immunising agent; this question also
at present has not been satisfactorily answered. The filtered gall was proved
not to be capable of giving the disease, and also does not protect from the
disease, but the matter which remains on the filter, even after being twice
washed with normal salt solution, while it does not communicate rinderpest,
yet protects the animal from the disease.

C.—A series of experiments was instituted by Kolle and Turner to ascertain
the amount and kind of action that immunising serum exercised over the
rinderpest virus outside the body, in order to gain some idea of how it pro-
duced its action when injected as a therapeutic agent into animals suffering

from the early manifestations of the disease or as a protective agent against attacks of the disease—in other words, to compare the action of the serum *in vitro* with its action *in corpore*, in the hope of ascertaining by this comparison some idea of its mode of action in the latter case. Mixtures of rinderpest blood and immunising serum were made of the following strengths :— (1) 0·5 per cent. of blood to serum ; (2) 1·0 per cent. of blood to serum ; (3) 2·0 per cent. of blood to serum ; and (4) 10·0 per cent. of blood to serum. These mixtures were kept in each case twenty-four hours in a cool place before being used. In Experiment I. 10 cc. of the 0·5 mixture were injected into two animals. The result was negative ; neither did the animals take rinderpest nor were they rendered immune, both succumbing eventually to an inoculation with rinderpest blood on the eighth day after the first experiment. In Experiment II. 20 cc. of the 1 per cent. mixture were injected into eighteen animals. The result was that seven had rinderpest and four died ; eleven of the animals showed no reaction, and were subjected to a second inoculation of rinderpest blood only. All of them had rinderpest and seven died. A considerable difference in the ultimate result, apparently dependent on the time allowed to elapse between the first and second injections, is noticeable in this experiment. Seven of the animals which had showed no reaction were injected for the second time seven days after the operation ; only three of these died, four recovering from the disease. Of the remaining four cattle which had been unaffected by the first inoculation, two were injected with virulent blood on the eighth day and two on the tenth day after the first operation ; all four died, thus apparently showing that the first injection had some power in fortifying the animals against the injection of rinderpest blood if this was performed within seven days. In Experiment III. 20 cc. of the 2 per cent. solution were injected into three oxen. In Experiment IV. 10 cc. of the 10 per cent. solution were injected into three oxen. In both cases all the oxen died from the disease.

These experiments showed that, working with ordinary unfortified serum and virulent blood *in vitro*, the virus was destroyed when present in small proportion—0·5 per cent. When the proportion was increased to 1 per cent. the virus appeared to be destroyed in about 60 per cent. of the cases, to be impaired in 20 per cent. of the cases, and to be undiminished in 20 per cent. of the cases treated. In higher proportions the virus was undiminished. Working with fortified serum similar results were obtained.

We may conclude, then, that *in vitro* the bactericidal action of the serum obtained from salted animals is small, and that the outside limit of its power does not exceed 1 per cent. of its volume of rinderpest blood even after standing twenty-four hours, but that it does exert a modifying influence on the poison is evidenced by the results of the experiments with 0·5 per cent. and 1 per cent. solutions. If now we take results, on the other hand, of experiments in which immunising serum was injected into animals already infected with the disease, we find that a dose of from 50 to 100 cc. of immunising serum is sufficient to overcome and counteract the effect of an injection of even from 100 to 200 cc. of rinderpest blood, provided the former were injected any time within twenty-four hours after the blood inoculation. A comparison of these results shows conclusively that the action of immunising serum on the virus of rinderpest *in vitro* is infinitely less powerful than when applied *in corpore*. In other words, its action on the poison outside the body is very different from its therapeutic power in the living organism ; evidently this difference in its action is brought about chiefly through the intervention of the living cells of the animal experimented on. Dr Kolle and Dr Turner suggest that the immunising material present in the serum injected is in an inactive modification, that it acts as a stimulus to the cells, probably nervous, and that these cells, again acting upon the injected material, convert it from an inactive into an active modification.

D.—With regard to the preparation of the serum, the first serum used was obtained from an animal which had recovered from rinderpest without any treatment, but the serum proved so weak that it was practically useless. The next step was to attempt to increase the power of the serum by injecting into the animal doses not exceeding 100 cc. of rinderpest blood. This improved the quality of the serum, as proved by the experiments of Dr Koch. Dr Kolle and Dr Turner were led by degrees to increase the doses of virulent blood up to 4000 cc., and they found that the power of the serum was thereby most decidedly increased. Experience convinced them, however, that animals which had survived a severe attack of the disease are by no means the best suited for the purpose of serum preparation, and they now prepare it from healthy animals which have been immunised by the simultaneous method of inoculation and afterwards fortified by successive inoculations of rinderpest blood. The greatest care is requisite in the examination of the blood used for fortifying animals, so as to ensure it being free from septic and other organisms, and in order to exclude as far as possible the possibility of it containing the germs of other diseases. The animals having been fortified in the way stated, they are bled in the usual way, and the serum is collected after the clot has formed or by means of a centrifugal machine. The serum has now to be tested, so as to gain an approximate knowledge of its strength. This is done by a series of simultaneous inoculations. Ten or twelve healthy animals are taken, their weights ascertained approximately by measurement to be 600 lbs. Each is then injected with the same quantity of virulent blood, and then, say, three of these are injected simultaneously with a dose of 15 cc. of fortified serum, three again with 20 cc., three others with 30 cc., and perhaps three others with 40 cc. The animals are then allowed to stand; food, water, and shelter only are provided; no treatment of any kind is attempted. From the results obtained the strength of the serum is judged. Suppose that some of the animals injected with 15 cc. die and that the others are severely ill, that those injected with 20 cc. have a mild attack, that those injected with 30 cc. do not all suffer, and that none of those injected with 40 cc. suffer, it would be considered that the dose of that particular batch of serum should be placed at 20 cc. for animals weighing 600 lbs. The serum is then mixed with carbolic acid in the proportion of 0·5 per cent. of the latter and bottled with proper precautions. It has been proved that it retains its strength for at any rate seven months, but for how much longer is uncertain, as sufficient time has not elapsed to show.

E.—As regards obtaining rinderpest blood for the simultaneous inoculation process, a difficulty arises in transporting it any distance from the fact that it rapidly loses its virulent properties, and any preservative added has a similar action. This difficulty is, however, to a great extent overcome by using the blood of living sheep as a means of conveying the poison to a distance. Although the latter animals themselves do not take the disease, still blood drawn from them from three to eight days after an injection of 100 or 200 cc. of virulent blood fatally infects oxen injected with it, and it has been proved that the strength of the virulent blood can be virtually disregarded in simultaneous inoculations.

NAGANA OR TSETSE FLY DISEASE.

At the request of the Colonial Office, the Royal Society of London appointed a Committee to co-operate with Surgeon-Major Bruce in his research upon nagana or tsetse fly disease, and to study nagana systematically in

ordinary laboratory animals, to investigate the life-history of the hæmatozoon discovered by Bruce, and, if posslble, to discover methods of prevention, cure, or immunisation.

The material for the observations was obtained in the first instance from the blood of a dog infected by the disease on the voyage from Africa, and brought to England in November 1896, by Dr Waghorn.

The Hæmatozoon of nagana has already been described by Bruce, and is closely allied to the *Trypanosoma* of Surra. The Committee had no opportunity of studying the latter disease. The parasite discovered and described by Rouget in a horse in Algeria is also similar. In English sewer rats (*Mus decumanus*) a *Trypanosoma* (*T. sanguinis*) is occasionally found, but this is quite distinct from the hæmatozoon of nagana, both in its morphological appearance and in its pathogenic effects.

Susceptibility.

Cats, dogs, mice, rabbits, rats, both sewer rats (*Mus decumanus*) and white and piebald rats (*Mus rattus*), are highly susceptible, and in these animals the disease proved fatal in every case of infection which was allowed to run to a close.

A single *hedgehog* inoculated was readily infected and died in seventeen days, so that this animal probably possesses a high susceptibility.

A single *donkey* was inoculated and was killed twelve weeks later, being then in a weak condition and near dying.

Two *horses* have been inoculated, one a strong and well-fed cart horse ("Russian"), which survived seven weeks, the other a rather old animal (*see* under zebra hybrids) which survived only eight days.

A *bosch-bok* has also been inoculated; it died seven months afterwards without showing any lesions. All the inoculations made from it proved negative.

Two hybrids of zebra and horse (♂ zebra and ♀ horse, and ♂ horse and ♀ zebra) and one hybrid of zebra and ass (ass ♂ and ♀ zebra) have also been inoculated.

The two former were infected by plunging a needle wetted with nagana blood beneath the skin; the latter received a dose of 1 cubic centimetre of the same blood. All of them died in about eight weeks. During the course of the disease they showed irregular rises of temperature, sometimes up to 41·6° C. Variations in the number of hæmatozoa were ascertained in the case of the horse hybrids; on some occasions they were abundant (66,000 per cubic millimetre). Whenever the donkey hybrid was examined at the earlier stage of the illness the hæmatozoa were found to be either scanty or absent. A horse which was inoculated as a control died in eight days, with very abundant hæmatozoa in its blood. There is no reason for supposing that the hybrids exhibit any more refractoriness than other horses or asses.

Koch [1] reports on attempts which he made to infect two Masai donkeys, and two crosses from Muscat and Masai donkeys. None of these showed any symptoms of the disease up to three and a half months, nor were hæmatozoa discovered in their blood at any time, although repeated examinations were made. Consequently there is no proof that these animals were really infected. Scratch inoculations sometimes, though rarely, fail; on the other hand, inoculations by puncture with a needle or by actual injection do not fail; Koch's animals were inoculated by the scratch method. It should be added that all his control infections were successful. He did not find that ordinary mules showed any immunity.

Guinea-pigs are susceptible to nagana under ordinary conditions, but, as a rule, the disease in them is more protracted than in rabbits, rats, mice, cats

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[1] " Reiseberichte," pp. 69 and 88.

or dogs, and even horses ; so that they are distinctly more resistant than these animals. In no instance, however, has recovery ensued after hæmatozoa have once appeared in the blood.

According to unpublished observations by Bruce upon the tsetse fly disease in South Africa, it appears that native *goats* and *sheep* are to some extent refractory, the disease, as a rule, running a chronic course (five months).

A *monkey* (*Macacus rhesus*) was also tried. It died in about two weeks in an advanced condition of pulmonary tuberculosis, but the presence of abundant hæmatozoa had been determined in the blood during life up to the time of death.

A *weasel* was injected. It showed hæmatozoa in its blood, and died a few days later, but death almost certainly was hastened by the effects of captivity.

Pigeons are the only *birds* which have yet been tried. The pigeons after inoculation did not show signs of the disease, nor was their blood infective. Bruce tried South African *hens* without success.

Young animals, if susceptible (kittens and puppies), as a rule have died earlier than adults, and while suckling they are still more highly predisposed ; young guinea-pigs, however, are comparable to older ones in their resistance.

The *fœtus in utero* of infected rabbits, guinea-pigs or rats, is not infected, although the mother's blood may contain a large number of hæmatozoa. The latter are to be found in the placenta, but not in the fœtal blood.

Duration of Disease in the Different Animals.

The lethal period varies somewhat in each species of susceptible animal. The duration of the disease appears to depend principally upon the individual susceptibility rather than on the mode of inoculation or the quantity of infective material introduced. Thus, of four rabbits inoculated in the same manner with the same material, three died on the 12th, 21st, and 24th days respectively, whilst the fourth was killed on the 41st day ; many similar instances could be cited. Nor does a larger quantity necessarily determine a more rapid death ; thus a rabbit which has received the whole blood of another rabbit containing numerous hæmatozoa may survive longer than the minimal lethal period for rabbits.

The ratio of the minimal to the maximal lethal periods is about 1 to 5 or 1 to 6 in rabbits and rats, and 1 to 9 in guinea-pigs. The number of other animals inoculated has not been sufficiently great to determine a satisfactory ratio.

In the experiments dogs survived an infection 14 to 26 days, cats 22 to 26 days, rats 6 to 26 days, mice 8 to 25 days, rabbits 13 to 58 days, and guinea-pigs 20 to 183 days ; the average duration being for dogs 18 days, for cats 24 days, for rats 12 days, for mice 13 days, for rabbits 30 days, and for guinea-pigs 50 days.

Since the commencement of these experiments, a large number of animals have been dealt with, and thus an extensive series of cross-inoculations has been carried out ; but it has been found that the duration of the disease is not dependent on the kind of animal from which the hæmatozoa are derived. No constant modification is, therefore, effected by passages, either in the direction of attenuation or of increased virulence. This statement is completely born out by Bruce's observations on wild animals, as well as African sheep and goats, for he found that the hæmatozoa of these animals were as infective as those obtained from highly susceptible animals, such as dogs.

Mode of Inoculation.

Inoculations have been made with the blood of an infected animal, subcutaneously, intravenously, or intraperitoneally, or by applying a minute and

often minimal quantity of infected blood to a superficial scratch. Rabbits have also been inoculated in the anterior chamber of the eye, and rats directly into a lymphatic gland.

Blood taken from diseased animals, although showing no hæmatozoa when examined microscopically, frequently proved to be fully infective, so that it appears that a single hæmatozoon, or at anyrate a very small number of them, successfully introduced, are capable of producing the disease. At present no method of graduating the dose appears possible, since a minute quantity is as effective as much larger quantities, though the lethal period may be somewhat prolonged. It is also possible that unrecognised forms are present in these cases, though in some instances where no hæmatozoa are found in simple films, they should be detected by means of centrifugalising the blood.

Successful inoculations have also been made with lymphatic gland, spleen, bone-marrow, aqueous humour, serous fluid, œdema transudation, and testicular juice.

The incubation period and the duration of the disease are not entirely dependent upon the number of hæmatozoa in the material injected, or the source of the infective material. Thus the hæmatozoa of lymphatic glands, etc., are as infective as those of the blood. The duration of the disease is not materially affected by the mode of inoculation adopted, and is about the same, whether the infection was brought about by subcutaneous, intravenous, or intraperitoneal injection, or by a superficial scratch.

Material taken from the bodies of animals twenty-four hours, or sometimes less, after death, is hardly ever infective, even when several cubic centimetres are injected, so that there is no evidence of a resisting or sporing form which survives in the tissues or blood of the dead animal, and is inoculable into other mammals. Putrefactive changes often set in with great rapidity in the bodies of animals dead of nagana.

Blood drawn from the living infected animal and kept *in vitro* in an aseptic condition, retains its infective power *at most* for three or four days, but this period is generally less. Complete drying also renders blood non-infective.

Blood heated to 50° for thirty minutes invariably becomes non-infective, even in large doses (such as 4 cc.), while when heated to 46° C. for half-an-hour it proved infective in one out of two cases, although apparently the hæmatozoa had become non-motile—at least no motile forms were detected under the microscope. But even in this case the lethal period was not prolonged.

Infection by feeding was attempted by means of a number of experiments. Sometimes it was successful, in most cases unsuccessful, so that the possibility of infection by the mouth seems to depend on accidental lesions about the mouth, nose, ears (in rats), or alimentary tract.

Of a number of rats fed on organs of nagana animals, only a few acquired the disease, and these invariably showed superficial lesions of the snout and ears, due to lice. When fed upon infective material, they bury their snouts in it as well as scratch their ears with their blood-stained forepaws. Furthermore, in the rats which acquired the disease through feeding, the cervical glands were always enlarged most, which proves that the hæmatozoa infection must have taken place in the head, for the primary infection travels by the lymphatics.

A cat fed repeatedly on soft tissues of the bodies of infected dogs and cats, and subsequently on the bodies of dead rats, died at a time corresponding by lethal period to an infection at the first meal on rats. Probably some splinter of bone caused a superficial lesion through which the hæmatozoa were enabled to enter.

One rabbit, fed carefully by means of a pipette with large quantities of

infected blood, never showed the slightest sign of the disease. Rouget also failed to infect animals by the mouth.

Two rabbits, into whose conjunctival sacs several drops of blood containing very abundant hæmatozoa, and a third rabbit whose eye was brought into contact with one of these, did not become infected.

A dog suffering from the disease did not infect her puppies during the last fourteen days of her life, nor did these puppies infect their foster mother (she-cat) after they had been inoculated.

Nor was the transmission of the disease through the mother's milk in guinea-pigs observed. Rouget alludes to a doubtful instance of infection by coitus in rabbits by means of the spermatic fluid.

The authors of the report do not believe that it is possible to infect an animal by feeding in the absence of superficial lesions, and in this respect they differ from Bruce, who seems to imply that the hæmatozoa can pass through the unbroken surface of the alimentary tract.

Symptoms and Course of the Disease.

Muscular wasting and *loss of power* are evident in all but the small animals. In rats, mice, and guinea-pigs they are but little marked or absent altogether. In the horse, dog, cat, and rabbit the wasting is very conspicuous. In the cat, dog, rabbit, and hedgehog there is marked loss of weight, amounting to 20 to 30 per cent.

Fever.—In most animals which have been examined, there is a smart rise of temperature about the time of appearance of hæmatozoa in the blood. (Horse, 41·5° C.; dog, 40° C.; rabbit, 41° C.; guinea-pig, not constant).

Paroxysms of fever are common in the *horse*, as has already been shown by Bruce. The temperature may rise to a considerable height (41·6° C.); the same is true of the zebra-horse hybrids. In a horse upon which daily observations were made, quick and sudden rises of temperature immediately followed an increase of the hæmatozoa in the blood. At the time of death there was marked pyrexia.

In the single *donkey* examined the temperature was generally raised throughout the course of the disease.

In *dogs* there is also fever, the temperature becoming subnormal on approach of death.

In *rabbits* pyrexia is common, and generally the temperature is elevated throughout the disease, but it may fall suddenly to normal. The temperature curve is always irregular, and no relation between the temperature curve and the hæmatozoal curve could be established.

In *cats* also the fever is well marked, the temperature falling quickly towards the end.

Œdema is common in certain animals, such as the horse, rabbit, cat, and dog, and is most marked about the head, legs, belly, or genitals. In smaller animals, such as rats and mice, it is not usual, and in guinea-pigs it has not been observed. In dense tissues, as the rabbit's ear, there may be a local œdema at the site of inoculation.

Rabbits exhibit a special tendency to œdema of the external genital organs. There is often great and progressive swelling of the prepuce or labia, as the case may be. The swollen parts often excoriate and become sore and covered by crusts, so that the animal is in a sorry condition.

Changes in the Eyes and Nose.—In cats, dogs, rats and rabbits turbidity of the aqueous humour, fibrinous plaques in the anterior chamber, and corneal opacities are occasionally observed. In rabbits a muco-purulent conjunctivitis is common, and this may be followed by an opacity of the cornea and a turbidity of the aqueous humour, which under such conditions shows hæmatozoa microscopically as well as leucocytes. Hæmatozoa have also been discovered in the conjunctival discharge in the earlier stages of the

disease. Vascular corneal ulcers sometimes occur in dogs, and the conjunctivitis of cats, dogs, rats and rabbits is frequently associated with œdema of the eyelids and face. In rabbits the eyelids and nose frequently become almost entirely closed up by the drying of the secretion; in the latter case they breathe with great difficulty, keeping their mouths open.

Anæmia.—Some degree of anæmia is always present, but it does not seem to be so extreme as to be the sole attributable cause of death, and points rather to a disturbance in the hæmatopoietic or the hæmatolytic mechanisms.

The number of red blood corpuscles steadily diminishes and nucleated red corpuscles (normoblasts) often appear, especially in rats. According to observations on rabbits the diminution of hæmoglobin is roughly proportional to that of the blood corpuscles.

Leucocytosis is not a constant feature, and when present is apparently due to the febrile temperature. An excessive leucocytosis, such as occurs in leukæmia, was never observed; 15,000 to 34,000 leucocytes being the highest numbers recorded per cubic millimetre.

Blood drawn from an animal seriously ill, when clotted, generally exhibits a marked buffy coat; the serum is often turbid, and may undergo secondary clotting.

Instead of forming rouleaux, the red corpuscles tend to clump into masses and to lose their outlines, especially when the anæmia is pronounced (rabbit, ass, and horse).

The serum of such blood, when mixed with normal blood of the same species of animal, causes the red corpuscles to clump together also.

The urine of infected dogs spectroscopically examined often shows an intense *urobilin* band.

Wounds do not heal well, and tend to break down and become septic, even though the operation has been performed with strict aseptic and antiseptic precautions. The hæmatozoa may be abundant in the discharge from the wounds. Many animals, especially dogs, are apt to become infected with pyococci and other bacteria in the later stages of the disease, even when the inoculated material has been proved to be free from bacteria. A spontaneous terminal bacterial infection may occur when the marasmus has reached a certain degree. This may accelerate death, and is probably fairly often the case in the naturally acquired disease, but bacteria are absent in uncomplicated cases of experimental inoculation.

A voracious appetite has not been observed in the infected laboratory animals; some animals refuse their food, and the stomach is not seldom empty after death.

Rats and guinea-pigs often exhibit convulsive or eclamptic seizures shortly before death, but otherwise guinea-pigs, rats, and mice show no symptoms of disease, except dulness in the later stages.

Transmission from one animal to another, without direct inoculation, has never been observed. Nor have instances of infection by coitus or through suckling been noticed, although large numbers of animals were used.

Morbid Anatomy.

In *rats* and *mice* exactly the same conditions may be observed. The most striking changes are :—

Enlargement of the lymphatic glands, the glands corresponding to the seat of inoculation being always largest. This observation is important, because from the relative size of the glands it is possible to determine the seat of infection. To this allusion has already been made, when the effects of feeding were discussed. The glands are generally red, congested, juicy, and œdematous; in a few instances hæmorrhagic extravasation has been observed. In some cases all lymphatic glands in the body are enlarged, in others a particular series only. If a rat be inoculated in the right thigh, the glands in

the left axilla and left groin suffer last. The spleen is much enlarged, with but few exceptions, and it is generally firm, friable, and dark coloured. The liver generally shows some enlargement, and may be fatty. Wasting of the muscles and atrophy of the fat is, as a rule, not well marked. Sub-pleural ecchymoses are sometimes present in the lungs, accompanied by a small amount of pleural fluid.

In *rabbits* the general enlargement of lymphatic glands is less noticeable. The spleen is generally enlarged. Petechial ecchymoses are rare. Fatty degeneration of the liver is always present, and muscular wasting is often extreme. Enlargement of testes has been observed.

In *dogs* muscular wasting is well marked, the animal being often reduced to a skeleton, but the fatty tissues are generally not much affected, except at the base of the heart, where the fat may undergo œdematous degeneration. The general enlargement of the lymphatic gland is well marked, and, as in the rat, the glands are œdematous and congested, yellowish, or even show hæmorrhagic extravasations. The spleen is also greatly enlarged, granular, firm, and friable. Pericardial effusion is common, and pleural effusion may be present. Sub-pericardial petechiæ and hæmorrhages occur frequently, sub-peritoneal occasionally, and sometimes also submucous in the intestines and stomach.

In *cats* wasting is pronounced, the glands are greatly enlarged, the spleen is also enlarged, the liver is slightly enlarged. Hæmorrhages beneath the pleura and pericardium have been noticed.

In *guinea-pigs*, which clinically often show no changes or symptoms at all, the morbid changes after death are not very well marked. The spleen is generally moderately enlarged, and occasionally even considerably; it is often very soft and rather pale. The lymphatic glands are distinctly, but as a rule only slightly, enlarged, those corresponding to the seat of inoculation being always the most affected.

Hæmorrhages have been observed in the lungs and in the stomach; serous effusions and œdema have not been noted.

In all these animals the bone-marrow is sometimes dark red in colour, at other times natural, or paler than it should be. In the shafts of the long bones the fat disappears and becomes replaced by "red" marrow.

In many cases an iron reaction has been obtained with the liver, spleen, and kidney (ammonium sulphide; and K_4FeCy_6+HCl).

Distribution of Hæmatozoa.

After a latent period of some days, hæmatozoa are invariably found in the blood at some time or other during the course of the illness.

Rats.—When the animal is inoculated with small quantities of infective blood, the latent period averages three to four days. When, however, a large number of hæmatozoa is inoculated into the peritoneal cavity, the parasites may be found in the blood even after a few hours.

When the hæmatozoa have once appeared in the blood, they are generally found therein to the end, gradually increasing in number till the blood literally teems with them. During the early stages of the disease, however, variations are frequently noted, inasmuch as an increase on one day may be followed by a marked decrease on the next. In a few cases they have even temporarily disappeared from the circulation for a day or two, but this is distinctly rare in rats and mice, although common in other animals.

At the later stages the hæmatozoa may amount to 2,000,000 to 3,000,000 per cubic millimetre.

Mice.—What has been said of rats applies also to mice.

Rabbits.—In these animals, after inoculations with minute quantities of blood, the parasites first appear in the blood in about eight days, about the same time as the pyrexial attack. They remain in the general circulation for

a day or two in small numbers; this is followed by a disappearance and reappearance for a variable number of days at irregular intervals. In the animals which have been systematically examined the hæmatozoa do not appear abundantly until towards the close of the disease; the largest number which has been estimated near the time of death has been 60,000 per cubic millimetre (compare rats and mice), but even at that time they may be scanty and difficult to find. They are also to be found in the fluid of the local œdema and discharge from wounds, conjunctiva, or genitals. Although hæmatozoa may be so scanty that they cannot be discovered by the microscope (sometimes even after centrifugalising), the animals show marked clinical symptoms. Their blood has often been proved to be infective.

Dogs —Early in the disease, from four to six days, the hæmatozoa may be absent from the blood, but observations on their presence during life in the lymphatic glands have not been made. Towards the end they become very numerous (100,000 to 300,000) per cubic millimetre). Variations in the number of hæmatozoa are common, but as a rule hæmatozoa are numerous throughout the disease.

Cats.—The latent period is about five days; then the hæmatozoa appear in the blood, and, with daily variations, quickly increase in number. The variations are sometimes remarkable; thus on one day the hæmatozoa may be extremely numerous, while on the next day they will have become scanty.

Horse.—In the first horse the latent period was seven days. The first appearance of hæmatozoa in the blood was followed by a sharp rise of the temperature. After the hæmatozoa once showed themselves they were generally scanty and often absent (the centrifuge was not used), but an appearance of the hæmatozoa in the blood was generally followed immediately by a paroxysm of fever. A few days before death, however, the number increased greatly, falling again to zero two days before death and being low at the time of death.

Guinea-pigs.—After a subcutaneous inoculation a few hæmatozoa will generally be found in the blood about the fifth to seventh day. They may then again disappear and reappear from time to time, to disappear again after a few days. This alternation may go on for weeks. Then suddenly the hæmatozoa become numerous and gradually increase, sometimes with irregular variations, till the blood is almost crowded, 200,000 to 500,000 per cubic millimetre being present. The guinea-pigs die, generally without showing any symptoms, except perhaps convulsive attacks a day or two before death.

In some cases no hæmatozoa have been found in the blood for over six weeks, although it has been examined daily. They then appeared in small numbers, and after remaining scarce for a week or so, suddenly and rapidly increased as the disease approached its fatal termination. It is, however, more common to find a few hæmatozoa about a week after inoculation, this being followed by a more or less prolonged period of absence.

In cases where the disease runs a less protracted course, the hæmatozoa become numerous about four weeks after the inoculation, when they are often present in large numbers; but, as in the case of other animals, the number of hæmatozoa may be very variable, being almost enormous one day and very considerably less, or even very small, the next. In a case where the guinea-pig had been bled before inoculation the disease ran a rather short course; hæmatozoa appeared nine days after the infection, rapidly rising in number to over 128,000 per cubic millimetre, the animal dying after twenty-two days. In a few cases where the lymphatic gland corresponding to the seat of inoculation was examined, hæmatozoa were found in the gland, whilst they were absent in the blood.

The animals may appear comparatively well whilst large numbers of

parasites are present in their blood and glands; this is especially the case with rats and guinea-pigs. On the other hand they may be seriously ill whilst the hæmatozoa are scanty in their blood; this obtains usually in rabbits, in which animals, as already stated, the glands do not become so much enlarged, and it is possible that the main effect of the parasites is borne by other organs. For instance, at times the bone-marrow has shown the presence of hæmatozoa, although search in other organs and in the blood proved negative.

After death in the various animals, hæmatozoa are to be found in most cases in the bone-marrow and spleen. The adult hæmatozoa may be common in these situations when but few are present in the blood; but this is not constant, for the reverse may be the case. Multiplication of the parasites certainly takes place in the lymphatic glands (rat) as well as in the infected area of connective tissue; it may also occur in the above-mentioned organs as well, and perhaps too in the blood.

The hæmatozoa are also found in the fluids of the serous cavities, at any rate when they are present also in the blood.

They have not been found in the intestinal contents, nor have they been seen in the urine, except in one case in which hæmaturia and submucous petechiæ of the bladder were present (rat).

Dead and non-motile forms may frequently be found in the circulation and in the lymphatic glands, when the disease is advanced. These are less defined and are ghost-like, being somewhat swollen in appearance; they are also generally in an extended condition.

Toxic Power of the Blood.

The fact that animals may appear to be well for days while hæmatozoa are abundant in their blood, suggests that the hæmatozoa do not secrete much, if any, specific toxin, and indeed so far no direct evidence has been obtained of a potent poison manufactured by the hæmatozoa, either by secretion or by chemical changes induced in the blood.

Fresh serum after filtration through Berkefeld filters, and blood or serum which had been kept for days in a sterile condition till the hæmatozoa had died, have had no specific toxic effects, even when large quantities have been injected into dogs, rats, or rabbits. Blood in which the hæmatozoa have been killed by exposure to 50° C. has had no more effect. The extracts of organs obtained from diseased animals have also shown no poisonous properties.

Immunisation and Cure.

Animals which have been repeatedly injected with blood or serum of nagana animals, such blood or serum having been previously freed from living hæmatozoa, either by filtration, heat, or by allowing it to stand for a week or longer, have not shown the slightest degree of an acquired immunity. Rats, rabbits, and dogs have been tested in this manner, but none of these animals have shown any diminution in susceptibility. Animals repeatedly injected with extracts of the organs of diseased animals have acquired no resistance. The blood of almost full-term fœtuses, prematurely born of highly diseased rabbits, has been tried, but without the slightest success in prevention or cure. The guinea-pig being a comparatively resistant animal, its serum has been used, but it also has no immunising action. Repeated inoculations of bile of diseased animals have been without preventive or curative effects, although in vitro bile, which is always free from hæmatozoa, rapidly destroys the hæmatozoa. Infective blood mixed with sufficient bile becomes non-infective, but confers no immunity. Previous inoculations with the hæmatozoon of the ordinary rat (T. sanguinis) have also been valueless. Sewer rats and white rats which have been repeatedly, but unsuccessfully, inoculated with the ordinary rat-hæmato-

zoon (*T. sanguinis*), and have been proved to be refractory to further inoculations with this hæmatozoon, have all contracted nagana when subsequently inoculated, and have died at the same time as control animals treated with an equal dose of infective blood.

Biology of the Nagana Hæmatozoa.

Rouget has failed to find forms corresponding to those described by Danilevsky in birds and by Shalashnikov in rats, and Lewis and others have been equally unsuccessful, while it is difficult to follow Lingard in his description of young forms. We have not succeeded in tracing a life history, and we are still in search of developmental forms, a task which at present occupies our special attention.

Most commonly in blood, etc., drawn from infected animals the forms described by Bruce are found. They are generally in active movement, and can sometimes be observed in locomotion with their flagellated end forwards, as Lewis described in the case of other hæmatozoa; in many cases they do not change their position by free swimming, but tend to fix themselves by one or other end to the coverslip or to corpuscles or cells in the specimen; they then exhibit more or less rapid oscillations, and may change their position by apparently drawing or pushing themselves in one direction or the other. Meanwhile the vibratile membrane waves rapidly and the protoplasmic body alters in shape, becoming thicker and shorter or thinner and longer; in the case of the English rat hæmatozoon free swimming is the rule; changes in the shape of the body like those of the nagana organism are not observed.

The nagana parasites vary considerably both in size and form; they may be long and pointed or blunt-ended and somewhat stouter; some individuals are short and thick with a short flagellum, their protoplasm being crowded with rounded granules. Still larger forms possessing more than one vibratile membrane are sometimes, though rarely, met with.

Especially in specimens taken from lymphatic glands, but also in specimens obtained from the blood, etc., there is a clear vacuole at the thick end; this does not become stained with staining reagents; it varies much in size in the different individuals, but there is no evidence that it is of a contractile nature.

By means of hæmalum or hæmatoxylin a nuclear body can be demonstrated in the middle of the parasite; it is usually oval, but may be more saddle-shaped. The protoplasm also contains a number of granules which stain with basophil reaction (methylene-blue, thionin, etc.); these are somewhat variable in number, being fewer in specimens in which the protoplasm is more refractive—*e.g.*, from lymphatic gland. These granules are distributed irregularly throughout the body of the parasite; they do not occur in the membrane or the flagellum. The chromatin spot situated close to the non-flagellated end in the *T. sanguinis* is not defined in the nagana *Trypanosoma*. The *T. sanguinis* also does not stain at all readily even by basic aniline dyes (dahlia, fuchsin, etc.), whilst that of tsetse disease is more readily coloured by these reagents.

Examples joined by the poles opposite the flagellum are common at times, but although they suggest perhaps conjugation, there is no evidence that this process does really occur. After prolonged observation no further changes have been noticed in these joined individuals.

In drawn blood or serous fluids the hæmatozoa eventually become motionless; this may occur rapidly, for instance in twenty minutes, but generally some motile specimens can be found after two to three days—sometimes, indeed, after as long as five or six. Within a corpse, the blood and organs become non-infective in about twenty-four hours.

Oval forms smaller than the ordinary hæmatozoa, with (or without) a short

flagellum, and often with a "beak" at the opposite pole, have been observed in the organs, but rarely in circulating blood.

Small rounded or ovoid bodies, about 1 to 2μ in diameter, hyaline, sometimes with a refringent chromatin spot or bipolar spots, or irregular (? amoeba-like) bodies, also sometimes with a chromatin spot and of the same size or rather larger, have been observed, especially in the lymphatic glands and bone-marrow, or spleen. It is possible that these are early stages in the development of the hæmatozoon. No forms have been seen at any time within the red blood corpusles.

Neither sporocystic nor larger distinctly amoeboid forms have been observed.

SEVENTH INTERNATIONAL CONGRESS OF VETERINARY SURGEONS, BADEN-BADEN, 9TH TO 14TH AUGUST 1899.

THE following gentlemen have undertaken to draw up reports on the subjects already announced for transaction and discussion by the Congress.

(a) *Precautionary measures against the spread of epidemic diseases in consequence of international trade in animals.*

Reporters.

Cope, Chief Veterinary Surgeon to the Board of Agriculture, London.

Dr Hutyra, Professor and Principal of the Veterinary Academy in Budapest.

Leblanc, Veterinary Surgeon for Epidemic Diseases, Member of the Academy of Medicine in Paris.

Vollers, Government Veterinary Surgeon in Hamburg.

(Swiss reporters are still wanting).

(b) *1. The prevention of tuberculosis among domestic animals.*

Reporters.

Dr Bang, Professor at the Veterinary College in Copenhagen.

Dr Siedamgrotzky, Privy Medical Councillor, Professor in the Royal Veterinary College in Dresden, District Veterinary Surgeon in the Kingdom of Saxony.

Dr Stubbé, Veterinary Inspector to the Ministry of Agriculture in Brussels.

(b) *2. The prevention of the use of the flesh and milk of animals suffering from tuberculosis.*

Reporters.

Butel, Veterinary-Surgeon to the Slaughter-House in Meaux.

De Yong, Government Veterinary Surgeon in Leyden.

Dr Ostertag, Professor at the Royal Veterinary College in Berlin.

(b) *3. The latest suggestions for an effectual meat inspection.*

Reporters.

Dr Edelmann, Superintendent of Meat Inspection in Dresden.

Kjerulf, Government Veterinary Surgeon in Stockholm.

Postolka, Imperial Official Veterinary Surgeon in Vienna.

(c) *The prevention of foot-and-mouth disease.*

Reporters.

Paul Cagny, Veterinary Surgeon in Senlis.
Cope, as above, of London.
Dr Dammann, Privy and Medical Councillor, Professor and Principal of the Royal Veterinary College in Hanover.
Dr Furtuna, President of the Veterinary Office in Bucharest.
Hafner, Councillor and Veterinary Referee of the Grand Ducal Ministry of the Interior in Carlsruhe.
Hess, Professor at the Veterinary School in Berne.
Lindquist, Professor and Principal of the Veterinary College in Stockholm (has not yet given a decided answer in the affirmative).
Dr Wirtz, Professor and Principal of the Veterinary College in Utrecht.

(d) *The prevention of swine fever.*

Reporters.

Leclainche, Professor in the Veterinary School at Toulouse.
Dr Lorenz, Grand-Ducal Medical Officer in Darmstadt.
Dr Perroncito, Professor at the Veterinary Academy in Turin.

(e) *The extension of veterinary instruction, especially by the establishment of institutions for experiments in diseases and by founding chairs of comparative medicine in colleges for veterinary surgery.*

Reporters.

Degive, Professor and Principal of the Veterinary College in Brussels.
Dr Kitt, Professor at the Royal Veterinary College in Munich.
Dr Malkmus, Professor at the Royal Veterinary College in Hanover.
Dr Nocard, Professor at the Veterinary College of Alfort, Paris, Member of the Academy of Medicine.
Dr Raupach, Councillor, Professor and . Head of the Imperial Veterinary Institute in Dorpat.
Dr Schütz, Privy Councillor, Professor at the Royal Veterinary College in Berlin.

(f) *Conclusion of the work of drawing up a universal anatomical nomenclature in veterinary medicine in accordance with resolutions passed by the Sixth Congress.*

Reporters.

Dr Ellenberger, Medical Councillor, Professor at the Royal Veterinary College in Dresden.
Dr Sussdorf, Professor at the Royal Veterinary College in Stuttgart.

(g) *Veterinary Officials.*

Reporter.

Dr Lydtin, Privy Councillor in Baden-Baden.

The majority of reporters have agreed to send in their reports by January 1899. The translation and printing of the reports will take about two or three months, though single reports may be ready during the first quarter of 1899.

In order that the gentlemen who wish to take part in the work of the Congress, or who take an interest in this work, may receive the reports and other publications of the Congress at the earliest possible date, it is requested that applications for membership shall be sent in at once or at the latest by 31st March next year. This is to be done by sending the Member's fee of 12s. to the *Filidle der Rheinischen Credit Bank in Baden-Baden.* Gentle-

men who become Members, even if unable personally to be present at the Congress, will receive by post, free of charge, all publications, including the general Report of the proceedings.   Those gentlemen who do not enter their names until the opening of the Congress will only receive the publications supplementarily.

The Committee of Management begs to draw attention to the fact that applications for apartments and rooms in boarding-houses can already be received by the Lodgings' Committee, Lichtenthalerstrasse 9 I Stock Baden-Baden.

Professor Noyer of Berne, General Secretary of the Sixth International Congress of Veterinary Surgeons, Mr Siegen of Luxemburg, Government Veterinary Surgeon, Mr Haas of Metz, District Veterinary Surgeon, President of the Veterinary Society of Alsace-Lorraine, and Mr Zündel of Mulhausen, Alsace, District Veterinary Surgeon, have kindly undertaken to act as interpreters from German into French and *vice-versa*.   The interpreter for English will not be decided upon until an adequate number of English-speaking members is announced.

The Committee of Management.

DR CASPER, Hoechst,
Secretary.
DR LYDTIN, Baden-Baden,
President.

BADEN-BADEN, *3rd November* 1898.

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